EXPIRATORY MUSCLE STRENGTH TRAINING AND DETRAINING: EFFECTS ON SPEECH AND COUGH PRODUCTION

By

SUSAN E. BAKER

A DISSERTATION PRESENTED TO THE GRADUATE SCHOOL
OF THE UNIVERSITY OF FLORIDA IN PARTIAL FULLFILLMENT
OF THE REQUIREMENTS FOR THE DEGREE OF
DOCTOR OF PHILOSOPHY

UNIVERSITY OF FLORIDA

2003

ACKNOWLEDGEMENTS

First and foremost I would like to thank my advisor and dissertation committee chair, Christine Sapienza, Ph.D. Her mentoring has helped me achieve more than I ever thought possible. The only way I know how to repay her for her dedication is to pass on the knowledge she has given to me and provide a similar supportive environment for my future students.

I would like to express my sincere appreciation to my dissertation committee members, W.S. Brown, Jr., Ph.D., Howard Rothman, Ph.D., and Paul Davenport, Ph.D. Their support and guidance throughout my entire doctorate program has been tremendous.

Several colleagues and fellow students deserve a note of appreciation for their efforts on various stages of this project as well. I would like to thank Rahul Shrivastav, Ph.D. for his help with writing an algorithm in MATLAB for some of the data analysis. I would also like to thank several of the undergraduate students that worked in our laboratory over the past year that dedicated their time to this project. These students include Michelle Vivaqua, Sara Lancashire, Jordan Kelgren, and Michelle Troche. I would also like to thank my lab colleagues over the years, Bari Hoffman Ruddy, Ph.D., Yaser Natour, Ph.D., and Judy Wingate for their support and friendship.

Finally, my love and thanks is given to my family for their support and encouragement throughout my entire academic career. I would also like to thank Bonnie and Philip Ankrim for being my "Gainesville Family." I will never forget their kindness.

TABLE OF CONTENTS

Abstract of Dissertation Presented to the Graduate School
of the University of Florida in Partial Fulfillment of the
Requirements for the Degree of Doctor of Philosophy

EXPIRATORY MUSCLE STRENGTH TRAINING AND DETRAINING: EFFECTS ON SPEECH AND COUGH PRODUCTION

By

Susan E. Baker

August 2003

Chair: Christine Sapienza
Major Department: Communication Sciences and Disorders

Reduced expiratory muscle strength compromises the necessary lung pressure for tasks such as speech and cough. It is known that expiratory muscle strength can be increased with strength training programs. However, there is limited research on the rate and magnitude of expiratory muscle strength increase, the physiologic changes in speech and cough following training, and alterations to these physiologic changes as a function of detraining.

Participants included 32 (20 females; 12 males) healthy individuals with an average age of 25.2 years. A multivariate repeated measures design was used in which measures of expiratory muscle strength, speech, and cough were obtained each week during an expiratory muscle strength training (EMST) program. The participants were divided into two training groups. Group I trained for four weeks. Group II trained for eight weeks. Maximum expiratory pressure (MEP) was used as the measure of

maximum expiratory muscles strength. Speech measures were the ratio of subglottal pressure (P_s) to MEP and intraoral pressure (P_0) to MEP, maximum P_s, and peak root mean square. Cough measures were maximum flow rate, compression time, and rise time. All measures were also obtained during an eight-week detraining period to document the rate and magnitude of the strength decrease as well as the loss of physiologic changes in speech and cough.

The results demonstrated a significant increase in MEP in both Group I and Group II, $p<.05$. A plateau in strength increase was found following the sixth week of training and stayed constant up until the eighth week of training. A significant decrease in P_s/MEP occurred in Group I, $p<.05$, but not in Group II. A significant decrease in P_0/MEP was found in both Group I and II, $p<.05$. No significant alterations in cough were noted with training except for a significant decrease in compression time in Group II, $p<.05$. MEP remained significantly above baseline at eight weeks post-training for both Group I and II, $p<.05$. These groups did not significantly differ in detraining rates. The results further indicate that investigation of EMST may be of interest in clinical populations which present with expiratory muscle weakness.

CHAPTER 1
GENERAL INTRODUCTION

Respiratory muscle training is a technique that has historically been utilized to increase the endurance and/or strength of the respiratory muscles for the purpose of improved ventilation. This technique has been studied in a variety of populations that present with resistive or obstructive diseases of the respiratory system. It has been hypothesized, and preliminary results suggest, that these increases in strength improve functions that require higher respiratory force such as speech and cough production.

Previously, whole body general exercise and fitness programs were used to target improvement in ventilatory function in patients with respiratory disease. A review of treatment outcomes from these programs was provided by Shaffer and colleagues (1981). This review revealed that very few of these studies demonstrated a significant increase in most ventilatory function parameters. While these programs demonstrated improvement in ventilatory endurance, these gains appeared to be due to an increased ability to use compensatory breathing patterns (Powers et al., 1997). Other training programs have used deep breathing exercises (Weins et al., 1999), abdominal muscle exercises (not associated with respiratory maneuvers) (Simpson, 1983; Vegeret et al., 1987), and abdominal weights to improve ventilatory function (Derrickson et al., 1992). All of these programs demonstrated limited success in significantly improving ventilatory function as they were not task-specific to respiration and/or lacked the ability to provide a sufficient load to the respiratory muscles to generate large increases in strength.

In 1976, Leith and Bradley demonstrated that respiratory muscle strength and endurance could be increased using exercises that were task-specific to the respiratory musculature. They used normocapnic hyperpnea to obtain increases in strength and endurance. This type of respiratory training requires the participant to ventilate to a higher degree over a period of 12 to 15 minutes by using a partial rebreathing system that maintains a constant $C0_2$ level. The presence of a higher level of $C0_2$ in the rebreathing system causes the individual to breathe more rapidly so that the excess $C0_2$ (hyperventilation) can be expelled. As a function of this training, a 55% increase in respiratory muscle strength was noted in healthy individuals after completing a five-week training program.

An important principle of skeletal muscle strength training is that the training maneuver should be specific to the targeted muscle group to gain the greatest effect. Leith and Bradley's training procedure was one of the first programs to specifically target the respiratory musculature at high loads. While this important work demonstrated the potential of respiratory muscle training as a treatment modality for increasing the strength and endurance of the respiratory muscles, it was not a practical training system as it required extensive monitoring of the patient in a laboratory setting.

Device driven programs are now predominately used to increase respiratory muscle strength. Resistance-based devices were used initially, which exposed the respiratory muscles to much higher loads than those obtained with abdominal or deep breathing exercises. Resistance training requires an individual to inspire or expire through a mouthpiece that provides a resistance to the flow of air. In the late 1980's, another respiratory muscle training technique emerged called pressure threshold training.

Pressure threshold training requires that the individual produce a specific amount of lung pressure into the training device before a one-way valve opens and allows air to pass through the mouthpiece of the device. The main difference between resistance and pressure threshold training techniques is that pressure threshold training is not flow dependent, allowing the training task to occur only when the respiratory muscles produce a sufficient lung pressure. Resistance training is flow dependent, in that the training task is completed when air flows through the device regardless of the lung pressure generated by the respiratory muscles (Gosselink & Decramer, 1994). It remains to be determined which of these training techniques is more effective. However, individuals are known to compensate during resistance-based training by decreasing flow, thus reducing the amount of muscle contraction necessary to complete the training task. Therefore, it is hypothesized that pressure threshold training has the potential to provide a greater training effect since it requires higher levels of muscle force in order to complete the training task at a specific load compared to the same load presented with resistance-based training. Because of this potential for a greater training effect, it is hypothesized that pressure threshold training will result in greater increases in respiratory muscle strength.

Respiratory muscle strength programs are implemented in much the same manner as limb muscle strength training programs. Typically an individual will be assigned a specific number of exercise sets to complete during a certain number of days per week. Training programs are generally prescribed and/or directed by a pulmonologist, allergist, physical therapist, respiratory therapist, or a speech-language pathologist. Evidence that pressure threshold training increases strength can be found in the work of Kellerman and colleagues (2000) who trained inspiratory muscles in healthy participants and O'Kroy

and Coasts' (1993), Sapienza and colleagues' (2002) and Suzuki and colleagues' (1995) work who showed strength increases in expiratory muscles of healthy individuals. Clinical applications of respiratory muscle training are described below.

Inspiratory muscle strength training (IMST) has been used with clinical populations who have difficulty moving air into the lungs due to obstructive or resistive disease processes. Strength training of the inspiratory muscles has been found to be successful in patients with chronic obstructive pulmonary disease (Grassino, 1989; Larson et al., 1988; Nield, 1999; Sonne & Davis, 1982), cystic fibrosis (Asher et al., 1982; deJong et al., 2001), asthma (Weiner et al., 1992; Weiner et al., 2000), upper airway obstruction (Baker et al., in press; Baker et al., 2003; Sapienza et al., 1999), and spinal cord injury (Sapienza et al., 2001).

While expiratory muscle strength training (EMST) has been studied far less than IMST, several investigations have pointed to its effectiveness in increasing expiratory muscle strength in individuals who either have difficulty moving air out of the lungs or who require higher expiratory drives for tasks such as coughing and singing. Increase in expiratory muscle strength following strength training in patients with skeletal muscle weakness has been linked to subjective reports of improved cough production (Gosselink et al., 2000; Smeltzer et al., 1996). Cough is a reflex response to the sensation of a substance such as mucus or a foreign object in the airway or is performed voluntarily. An adequate cough relies heavily on the generation of sufficient expiratory lung pressure in order to expel the substance or object from the airway. Pulmonary complications such as aspiration are a common risk factor in patients with skeletal muscle weakness (Gooselink et al., 2000; Szeinberg et al., 1988).

Sufficient expiratory lung pressure is also necessary to initiate and maintain vocal fold vibration during speech and singing. While the necessary expiratory lung pressure for most conversational speech tasks is approximately four to six cmH_20, individuals with significant weakness of the expiratory muscles may have difficulty maintaining this consistent lung pressure over extended periods of time. Significant reductions in lung pressure result in decreased vocal intensity, decreased fundamental frequency, and overall reduction in vocal quality (Ishikki, 1964; Netsell, 1969; Scherer, 1991; Titze, 1994). Singing tasks or very loud speech tasks may require lung pressures up to 20 cmH_20 requiring more force from the expiratory muscles. Improvements in expiratory lung pressure generation may prove to enhance daily function for an individual with skeletal muscle weakness and/or enhance the performance of a vocalist (Hoffman Ruddy et al., 2001).

While much research has been completed in the area of respiratory muscle strength training, many of the existing studies lack consistency in the intensity and duration of the training protocols. Due to this lack of consistency, it is difficult to draw general conclusions as to the precise response of the respiratory muscles to strength training in regards to rate and magnitude of strength increase over time. Additionally, very few of the existing studies have followed participants post-training to examine decreases in strength over time. Knowledge of effective training lengths as well as detraining rates of the respiratory muscles is important for developing research and clinical protocols with patient groups in the further study of this rehabilitation option.

Additionally, respiratory muscle training studies have been criticized for failing to demonstrate objective functional outcomes as a result of training. The most common

outcome parameters included in these respiratory training studies are indices of increased strength. Most often the only reports of improvement in tasks requiring higher ventilatory effort (speech and cough) are subjective comments provided by the participant or demonstrated on subjective rating scales such as "ease of breathing" or "effectiveness of cough."

Therefore, a methodological examination of the response of the respiratory muscles to varying durations of training and the cessation of training was performed with healthy participants. This study focused on expiratory muscle strength training due its potential for rehabilitating patients with voice disorders and dysphagia. This study will be presented in the format of four separate articles in the following chapters. The study presented in Chapter Two examines the influence of increases in expiratory muscle strength on measures of sound production. The study presented in Chapter Three examines the influence of increases in expiratory muscle strength on measures of cough production. A study of the magnitude and pattern of expiratory muscle strength changes over time is presented in Chapter Four. Finally, a study of the rate and magnitude of decreases in expiratory muscle strength during a detraining period is presented in Chapter Five. Chapter Six provides a summary of the four studies as well as a discussion of future research in EMST.

CHAPTER 2
SPEECH PRODUCTION WITH INCREASED EXPIRATORY MUSCLE STRENGTH

Introduction

The expiratory muscles play a critical role during speech production by providing an active force when the elastic recoil forces of the lungs are no longer able to generate the necessary lung pressure for vocal fold vibration (Hixon, 1973; Hixon et al., 1976; Hixon & Weismer, 1995). The average maximum amount of pressure that can be developed with expiratory muscle force in healthy young individuals is approximately 233 cmH_20 in males and 152 cmH_20 in females (Black & Hyatt, 1969). Subglottal pressure (P_s) is a small fraction of the maximum pressure and is defined as the air pressure beneath the vocal folds that initiates and maintains vocal fold vibration (Titze, 1994). For comfortable effort conversational speech, P_s is generally four to six cmH_20 (Scherer, 1991; Schneider & Baken, 1984; Smitheran & Hixon, 1981). Maximal expiratory muscle force is not typically required during comfortable effort conversational speech. In fact, less than 5% of the maximal expiratory muscle force is actually used to produce speech as most speech is produced in a mid-lung volume range. Consequently, expiratory muscle force is rarely needed because the passive elastic recoil force of the lung-thorax unit generates enough pressure to complete the task. For loud speech and singing tasks that require a greater P_s demand (10-25 cmH_20), the need for greater expiratory muscle force increases (Hixon, 1987). Likewise, there are individuals, who because of a disordered physiological system, are unable to produce the necessary

respiratory forces to inspire to large lung volumes, and thus the passive recoil force is compromised. These individuals may also have weak expiratory muscles, and when speaking tasks require additional force to generate or maintain P_s, they are unable to do so. This occurs in cases of neurodegenerative diseases such as Parkinson's disease due to rigidity of the intercostal muscles and in spinal cord injury due to muscle weakness. Inadequate P_s results in severely decreased vocal intensity, alterations in fundamental frequency, and shortened spoken utterances, all of which are detrimental for effective verbal communication (Isshiki, 1964; Scherer, 1991; Titze, 1994).

Reduced respiratory muscle strength has also been implicated for patients with multiple sclerosis (Gosselink et al., 2000), amyotrophic lateral sclerosis (Polkey et al., 1998) and muscular dystrophy (Szeinberg et al., 1988). Much of what is known about respiratory muscle strength weakness indicates reduced pulmonary function and cough production, yet little exists about quantifiable deficiencies in P_s and intraoral pressure (P_0). Intraoral pressure is the mouth pressure developed during speech and is a function of adequate expiratory force generation, vocal fold valving and articulatory postures (Netsell, 1969). Consequently, speech dysfunction can include decreased P_s and P_0 when expiratory force is limited. While P_s is directly related to inspiratory and expiratory muscle strength, P_0 can be related to reduced respiratory strength as well as poor muscle control and strength of the articulators (Solomon & Hixon, 1993). In fact, the Lee Silverman Voice Therapy program, the most widely utilized speech therapy technique for Parkinson's Disease, heavily focuses on increasing the expiratory drive for speech production (Ramig et al., 2001) but also aims to increase the range of articulator motion including laryngeal adduction.

It is known that individuals who are required to speak or sing during high levels of exertion have breathlessness and a decreased ability to produce long phrases (Hoffman Ruddy et al., 2001) as well as the perception of increased effort when performing high ventilatory tasks (Bailey & Hoit, 2002). It is speculated, due to the known function of the inspiratory and expiratory muscles during singing and speaking tasks, that these alterations in speech and song production may be due to fatigue of both inspiratory and expiratory muscles. It is also known that in order to sing, P_s must be increased to levels above the typical comfortable pressure range in order to meet the loudness and duration demands. When choreography is added to the performance, the physical load on an individual increases, demanding increased ventilation. The inspiratory muscles can fatigue because of the need for frequent and large inhalations for singing or speech phrases. Fatigue of the expiratory muscles may occur, as the primary abdominal muscles, which play a role in torso stabilization, are active during high levels of movement (i.e., dancing). Over time, fatigue of these muscles can lead to greater difficulty in achieving sufficient lung pressure for loud speech and lengthy singing phrases.

Increasing the strength of the expiratory muscles may enhance an individual's ability to generate and maintain P_s because the expiratory driving force is larger. In fact, several groups of researchers have implemented expiratory muscle strength training (EMST) protocols with both healthy individuals and clinical populations for the purpose of improving speech production and singing. These studies provide preliminary results that reveal variable outcomes with regard to speech parameters.

The outcome of a four-week EMST program implemented with eight theme park vocal performers indicated an 84% increase in expiratory muscle strength (Hoffman

Ruddy et al., 2001). These performers trained five days per week at 75% of their maximum strength. These performers reported a significant decrease in breathlessness during singing tasks with choreography and a significant increase in phrase duration.

Another study described the outcome of a six-week EMST training program with ten children with general hypertonia without respiratory and/or neuromuscular disease (Cerny et al., 1997). This study revealed a 69% increase in maximal expiratory pressure (MEP). Both P_s and vocal intensity increased following training. The participants wore a CPAP mask with a spring-loaded valve in the expiratory port. The resistance through the expiratory port began at 2.5 cmH_20 for all participants and was increased up to 7.5 cmH_20 for participants who were able to tolerate the higher resistance with training. The participants completed this breathing task 15 minutes a day, five days per week for six weeks.

A six-week EMST program was also used as a treatment modality in a randomized clinical trial of voice therapy for teachers with vocal disturbances (Roy et al., 2003). Expiratory muscle strength training was compared to resonant voice therapy and the use of a voice amplification system in the classroom. Laryngeal compensatory hyperfunction exists with this population, much like the singers, because of their high occupational voice demand. This compensatory strategy can further exacerbate vocal fold lesions. The participants trained at 80% of their maximum strength, five days per week. While expiratory muscle strength did increase with the EMST program, it did not significantly change the main outcome parameter compared to the other treatment modalities. The main outcome parameter in this study was the pre- and post-training score on the Voice Handicap Index (VHI) (Jacobson et al., 1999). The VHI is a

questionnaire that addresses quality of life in regards to vocal quality. This finding supported EMST training as beneficial but suggested that it may be of more benefit to those persons who have significant muscle weakness or who require very high expiratory drives such as during singing. While teachers often speak continuously throughout the day, the necessary P_s for their speech does not require significant expiratory muscle activation.

Further study of the alterations in speech/voice production that may occur with increased expiratory muscle strength is warranted at this time. An EMST protocol was implemented with healthy participants to examine the physiologic parameters that change with training as well as other key variables related to speech production. It was hypothesized that specific parameters of speech production such as maximum intensity and P_s during a dynamic range task would increase as expiratory muscle strength increased. It was hypothesized that measures of P_s and P_0 during comfortable speech tasks would not change with increased expiratory muscle strength, however, the total pressure generating capability (MEP) would increase. Therefore, it was hypothesized that there would be a decline in the ratio of P_s/MEP and P_0/MEP with training. Changes in these ratios in healthy participants will serve as a model of comparison for individuals who speak in high effort situations or have expiratory muscle weakness. Participants in this study were enrolled in either a four-week or eight-week training program. It was hypothesized that greater strength gains would be achieved with the longer training program and thus lead to greater alterations to the speech parameters of interest.

Methods

Participants

Thirty-two healthy participants completed this study. Twelve participants were males. The age of the males ranged from 19 to 32 years with an average age of 24.6 years. Twenty participants were females. The age of the females ranged from 19 to 48 years with an average age of 25.6 years Participants were recruited from the Gainesville, Florida, area.

All participants' MEP values were within a normative range for age and sex (Black & Hyatt, 1969). Pulmonary function testing was performed as a screening measure. All participants' forced expiratory volumes in the first second (FEV_1) and forced vital capacity (FVC) measures were within normal limits (American Thoracic Society, 1987; See Table 2-1). All participants had a negative history of chronic and acute cardiac disease, upper respiratory infection, pulmonary dysfunction/disease, neuromuscular disease, immune system disease, vocal disturbances (chronic or acute), obesity, and no history of smoking within last five years (tobacco or recreational drugs).

Only those participants who were able to maintain their current level of physical activity (including both aerobic exercise and weightlifting) throughout the entire training period were recruited. Participants were specifically asked to report any significant changes in their activity throughout their participation in the study with regards to intensity and frequency of exercise. An example of a significant change was defined as a sedentary person beginning to exercise two to four days per week. Participants were discontinued in the study if they made a significant change in activity level as described

above. Extreme athletes such as marathon runners and competitive weightlifters were also excluded.

Measures

Maximum expiratory pressure

Expiratory muscle strength was measured indirectly as MEP at the mouth. The measurement apparatus consisted of a mouthpiece connected to a pressure manometer by 50 cm of 2 mm i.d. tubing with a 14-gauge-needle air-leak. In order to measure MEP, the participants' nose was occluded with nose clips. After inhaling to total lung capacity, the participant placed his or her lips around the mouthpiece and blew out as forcefully as possible. Three repeated measures were taken with a one-minute rest between trials, until the measurements obtained were within 5% of each other. The average of these three values was used.

P_s/MEP and P_0/MEP

Estimated P_s was obtained from a syllable train (/pa/) completed at a comfortable loudness level. The pressure between two oral air pressure peaks was linearly interpolated during a syllable train of alternating voiceless plosives and voiced vowels, making it possible to estimate the P_s during the voice vowel segment. Participants repeated the syllable /pa/ seven times on one breath at a rate of 1.5 syllables per second (Lofqvist et al., 1982; Netsell & Hixon, 1978; Smitheran & Hixon, 1981). The middle five /pa/ syllables were measured from the syllable train.

Intraoral pressure (P_0) was defined as the pressure within the oral cavity during the production of the stop segment /p/ produced in the initial position of /papa/ produced during the reading of the *Papa passage* (Sapienza & Stathopoulos, 1995) (Appendix).

The passage consists of two paragraphs of 12 sentences (134 words) of varying length and phoneme representations with emphasis on the low vowel /a/ and voiceless stop /p/). The syllable repetition and the reading were produced at a comfortable effort level. Intraoral pressure was collected by inserting a small pitot tube (2 mm diameter) into the oral cavity between the lips and behind the front teeth. A pressure transducer (Glottal Enterprises, PTL-1), low pass-filtered at 30 Hz, was used to sense the air pressure. The pressure transducer was calibrated at 5 cmH_20 for each participant with a pressure calibration unit (Glottal Enterprises MCU-4). The pressure transducer was connected to an amplifier (Glottal Enterprises, MS100-A2). The amplifier was connected to a Power Lab/4SP data acquisition system (ADInstruments). Chart 4 for Windows (ADInstruments) was used to record the signal. Maximum pressures during the production of syllable trains were measured using this software program.

The ratio of P_s to MEP was calculated for each participant to determine the percentage of P_s generated relative to the maximum amount of pressure generating capability of the individual. The ratio of P_0 to MEP was calculated as well to determine the percentage of P_0 generated relative to the maximum generating capability of each individual. A decrease in these ratios reflects a smaller percentage of the pressure capacity used to produce speech. The P_s/MEP and P_0/MEP ratio served as an index for documenting changes in muscular strength for speech production.

Maximum P_s

The participants were asked to complete a dynamic range task in which they produced the syllable /pa/ from their softest to their loudest intensity level. Subglottal pressure was estimated from P_0, which was obtained in the manner described above. The

measure of maximum pressure during the final /pa/ of the dynamic range task was recorded.

Peak root mean square (RMS)

Peak RMS was defined as the rate of energy flow per unit area (or the sound power per unit area of the waveform) during the final, most intense /pa/ of the dynamic range task. Peak RMS was measured from an acoustic signal collected by a cardioid head-set microphone placed 2 cm from the right corner of the participant's lip. The microphone was calibrated in dB SPL (re: 0.0002 dynes per cm2) for the 2 cm mouth-to-microphone distance using an 90 dB pure-tone signal at 500 Hz. All acoustic signals were recorded using Cool Edit software (Syntrillium) and peak RMS was calculated using an automated algorithm programmed with MATLAB 6.2 .

Training Procedures

Expiratory pressure threshold trainer

An expiratory pressure threshold trainer was used to complete the EMST program. This cylindrical device consists of a mouthpiece and a one-way spring-loaded valve (Figure 2-1). The valve blocks expiratory airflow until a sufficient threshold pressure is reached to overcome the spring force. To achieve this threshold pressure, the participant breathed out with increased expiratory effort. As long as the threshold pressure is maintained, air flows through the device. The device contains an adjustable spring, which allowed the required threshold pressure to be increased.

Training protocol

Each participant was assigned to one of two training groups: a four-week training group or an eight-week training group. Each of the groups will be referred to as Group I

and Group II, respectively. Two training lengths were utilized in this study to examine the effect of greater training durations on speech parameters. The participants in each of the two groups were matched for sex due to expected differences in male and female strength gains. Males typically demonstrate greater increases in skeletal muscle strength in response to training programs (Ivey et al., 2000; Lewis et al., 1986). Likewise, greater increases in MEP were noted in males compared to females in a previous study that utilized pressure-threshold training in healthy participants (Sapienza et al., 2002).

Group I. These participants completed a pressure threshold expiratory training program for four weeks. As stated previously, the participants' MEP was measured at the initiation of the study and at the beginning of each subsequent training week. The threshold pressure was set at 75% of the participants' MEP at the time of measurement. The participants performed expiratory breathing exercises with the pressure threshold trainer five days per week. The training session consisted of five sets of five breaths. Each training breath lasted two to three seconds. The participant completed the training sessions throughout the week independently at home.

Group II. These participants completed the same program described for Group I, however, they trained for eight weeks.

Detraining. Both groups were followed for eight weeks after the completion of the training program. All of the dependent measures (MEP, maximum P_s, P_s/MEP, P_0/MEP, and peak RMS) were obtained again at four weeks post-training and eight weeks post-training.

Compliance

Participant compliance was acknowledged through participant education on the use of the device, mid-week contact by the investigator, and completion of a training log. Participants were provided with written and verbal instructions for the use of the device.

Statistical Analyses

The primary statistical method that was used to examine treatment differences with respect to the change from baseline scores across the two treatment groups for MEP, maximum P_s, P_s/MEP, and P_0/MEP was a multivariate repeated measures analysis of variance (MANOVA). Training week was the within-subject factor, sex was the between-subject factor. A separate MANOVA was performed for Group I and Group II because of the difference in the number of within-subject factor levels (four-weeks vs. eight-weeks). Significant differences at alpha'=0.05 were tested using univariate comparisons. Missing values in the data set were replaced using a linear trend function for the predicted values. There were a total of 33 missing values in the data set which represents less than 2% of the total data set. A univariate repeated measures analysis of variance (ANOVA) was completed for the peak RMS as this variable is an acoustic parameter which has little conceptual relationship to the other primary dependent variables. Planned simple contrasts were utilized to examine differences between dependent variables to the baseline measures across the weeks of training.

Results

A significant main effect was found for the within-subject factor of week for both Group I and II. (Table 2-2). Univariate tests for each of the dependent variables in Group I revealed a significant effect for all of the dependent variables. Univariate tests for each

of the dependent variables in Group II revealed a significant effect for MEP and P_0/MEP, but not for P_s/MEP or maximum P_s (Table 2-3).

A main effect for the between-subject factor of sex was found in Group I $F(4, 11) = 9.295$, $p < .05$ and II $F(4, 11) = 1.248$, $p < .05$. Univariate tests for each of the dependent variables revealed a significant difference between males and females for MEP in both groups and maximum P_s in Group I (Table 2-4).

Maximum Expiratory Pressure

Means and standard deviations as well as percent increase from baseline for MEP across the training and detraining period are in Tables 2-5 and 2-6. Figure 2-2 and 2-3 demonstrate the change in MEP over the training and detraining periods. Given the significant univariate results for MEP, analyzing the simple contrasts further helped interpret the effect. Contrasts showed that the baseline MEP was significantly different at the final week of training for both Group I and II. As well, significant differences between baseline MEP and MEP at the end of the fourth week and eighth week of the detraining period were found for both training groups. (Table 2-7).

P_s/MEP

Means and standard deviations as well as percent change from baseline for P_s/MEP across the training and detraining period are presented in Tables 2-8 and 2-9. Figure 2-4 demonstrates the change in P_s/MEP over the training and detraining periods for Group I. As the univariate analysis revealed a significant effect for P_s/MEP in Group I, planned simple contrasts for the within-factor of weeks were performed, indicating that baseline P_s/MEP was significantly different at the final week of training (week four).

These contrasts revealed the baseline P_s/MEP was significantly different at the end of the fourth week of the detraining period, but not by the eighth week (Table 2-10).

P_0/MEP

Means and standard deviations as well as percent change from baseline for P_0/MEP across the training and detraining period are presented in Tables 2-11 and 2-12. Figure 2-5 and 2-6 demonstrate the change in P_0/MEP over the training and detraining periods. As the univariate analyses revealed a significant effect for P_0/MEP in both Group I and II, planned simple contrasts for the within-factor of weeks were performed indicating that baseline P_0/MEP was significantly different at the final week of training for both groups (Table 2-13). These contrasts revealed significant differences between baseline P_0/MEP and P_0/MEP at the end of the fourth week of detraining and eighth week of detraining for both groups as well.

Maximum P_s

Means and standard deviations as well as percent increase from baseline for P_s/MEP across the training and detraining period are presented in Tables 2-14 and 2-15. As the univariate analyses revealed a significant effect for maximum P_s in Group I, planned simple contrasts for the within-factor of weeks were performed. These contrasts revealed only a significant difference between baseline maximium P_s and the eighth week of detraining (Table 2-16).

Peak RMS

Means and standard deviations as well as percent increase from baseline for peak RMS across the training and detraining period are presented in Tables 2-17 and 2-18. The repeated measures ANOVA performed for the variable peak RMS for Group I

revealed no significant main effect across the training weeks. The repeated measures ANOVA performed for Group II revealed a significant main effect $F(6,15) = 3.426$, $p < .05$. Planned simple contrasts revealed a significant difference only between the baseline peak RMS and peak RMS at the fourth and eighth week of detraining (Table 2-19). No significant differences between males and females were found in Group I and II.

Trainer limitations

Six of the male participants (three in Group I; three in Group II) achieved a MEP above 200 cmH_20 before the completion of the training program. The trainer was set to reflect 75% of the participants' MEP obtained each week. The maximum pressure threshold setting on the trainer used for this study was 150 cmH_20. Those participants who achieved a MEP above 200 cmH_20 during the training program continued to train with the trainer set at 150 cmH_20, however, this was at a pressure threshold less than 75% of their MEP. So even though these participants had achieved a MEP greater than 200 cmH_20, the training thresholds were unable to be raised in order to protect the participant from any potential risks that could have occurred when generating expiratory pressures above 200 cmH_20. Tables 2-20 and 2-21 present data from only those male participants who were able to train at 75% of their MEP throughout the entire training program.

Reliability

A second investigator measured approximately 10% of all the dependent variables except peak RMS as this measure was automatically extracted using a MATLAB algorithm. Pearson r correlations were used to determine the reliability between the measurers as well as t-tests to test for differences between the data sets. The Pearson r

correlation results are in Table 2-22. Strong correlations were found with all data sets and no significant differences in the data set were found according to the t-tests $p < 0.05$.

Additionally, the primary investigator measured approximately 10% of all the dependent variables expect peak RMS again for intra-measurer reliability. Pearson r correlations were used to determine the reliability as well as t-tests for differences between the measures. The Pearson r correlation results are in Table 2-23. Strong correlations were found will all data sets and no significant differences were found between the data sets according to the t-tests $p < 0.05$.

Discussion

The results of this study revealed a significant training effect in MEP in both Group I and II. The average increase from baseline across both groups was 45%. Other studies utilizing expiratory pressure threshold training have found similar positive increases in MEP occurring at about four weeks of training. The current study further supports that EMST is effective in increasing expiratory muscle strength. It also suggests that EMST impacts speech production due to the expiratory muscles' ability to generate increased respiratory pressure. During most conversational speech tasks, the expiratory muscles typically play a passive role and the work for pressure generation is completed by the release of stored elastic energy from the lung-thorax unit as well as a checking action from the muscles of inspiration (Hixon, 1987). The benefits of increasing MEP occurs when a speech task is performed at a lung volume where passive recoil no longer meets the subglottic pressure demand for phonation. When this occurs, expiratory muscles activate to increase the force on the lungs and increase pressure. This greater demand for expiratory muscle activation occurs during long speaking, singing phrases of

long duration, or any speaking situation where the initiation of the task is below about 60% of vital capacity (Hixon, 1987). For individuals who have skeletal muscle weakness or are speaking during high levels of exertion, the relationship of lung volume and the need for muscular work is different than what exists for a healthy individual. The ability to provide an increase in the force output and efficiency of the expiratory muscles has the potential for diminishing the perception of physiologic work or effort during speaking as well as substantially impacting the patients' ability to more effectively produce speech.

The study of the perception of effort and work during speech production is limited at this time. The concept of effort and work during speech production is highly pertinent to individuals who because of illness or demanding speaking situations, frequently report dyspnea or breathlessness during speech production. In a study by Bailey and Hoit (2002), chest wall kinematics and the perception of breathing difficulty was examined in healthy participants in a high effort speaking situation. This high effort speaking situation was simulated by having the participant breathe in the presence of high $C0_2$ levels. These participants only placed inspirations at inappropriate linguistic markers (i.e., not at a sentence, phrase, or clause boundary) 7% of the time indicating that they had little alteration in their speech patterns. However, they were queried for subjective comments regarding their difficulty breathing following the testing. The participants indicated that they felt breathless and that their work for breathing during speech had increased.

The etiology of breathlessness or dyspnea is complex in that it is influenced by multiple factors. In fact, it has recently been argued that the term dyspnea encompasses at least two distinct respiratory sensations: air hunger and increased work or effort for

breathing (Lansing et al., 2002). Mechanoreceptors and chemoreceptors located in the airways as well as the respiratory muscles are afferent sources for central breathing control centers in the medulla and higher central nervous system centers providing information that influence respiratory sensations (i.e., $PaC0_2$ and PaO_2 levels, lung volumes, and respiratory muscle activation). It could be argued that both air hunger (generally caused by high $C0_2$ levels) and increased effort are heightened during challenging speaking/singing tasks.

Research demonstrates that speakers are able to tolerate a small amount of metabolic imbalance to preserve linguistic markers in speech production (Mitchell et al., 1996). Metabolic imbalances during speech production are typically characterized by dropping to lower levels of vital capacity to finish a phrase appropriately creating a slight increase in $PaC0_2$ levels. Research also demonstrates that speakers employ compensatory breathing patterns during speech to reestablish $PaC0_2$ and $Pa0_2$ levels in high effort speaking situations (Bailey & Hoit, 2002). Therefore, it is assumed that dyspnea reported during speech production is in part due to air hunger. However, when an individual is required to ventilate under demanding conditions, the perception of physiologic work increases since work of breathing is dependent on the mechanical variables such as rate of respiration and tidal volume (Bartlett et al., 1973). Greater motor output is necessary to the expiratory muscles in order to maintain appropriate P_s and P_0 during speech. This increased activation from the motor cortex increases the perception of effort on a cortical level. It is hypothesized that EMST can alter neural and contractile properties of the expiratory muscles resulting in increased force output and efficiency. These alterations to expiratory muscles include increased motor unit recruitment and improved

synchronization of motor unit firing (Sale, 1988) as well as an increase in contractile proteins (myosin and actin) and type II fibers (fast-twitch) hypertrophy. Muscle spindles located in the expiratory muscles provide sensory information to the central nervous system regarding the length and contraction of these muscles. The increased efficiency of these muscles should then result in a decreased perception of effort at the central respiratory centers and further on at the cortical level.

Participants in this study were not queried regarding their change in perception of effort during speech production, as the experimental speech tasks were not strenuous enough to elicit a response. A questionnaire regarding perceived effort after EMST was used with the theme park performers previously described (Hoffman Ruddy, 2001) and these participants did report a decrease in effort and perceived breathlessness during singing tasks following the EMST program. Continued investigations between perceived effort and alterations in P_s/MEP and P_0/MEP would be useful in individuals with expiratory muscle weakness and individuals who are required to speak/sing at high effort levels.

Decreases were in fact found in P_s/MEP and P_0/MEP in this study. This decrease indicated that the participants were using less of their total pressure generating capability for speech production. Subglottal pressure and P_0 remained fairly consistent throughout the training period for all participants, while MEP increased. This subsequently reduced the ratio. Maintaining P_s and P_0 while increasing MEP was not surprising for this group of participants given that they were healthy. The decrease in P_s /MEP was significant in Group I, but not for Group II. This finding was surprising as MEP increased significantly in both groups. One possible reason for the lack of significant difference between Group

I and II is a slight inconsistency in P_s over the training weeks, not related to increased expiratory muscle strength, but rather variability in individual participant performance. The decrease in P_0/MEP was found to be significant across the training period for both Groups I and II.

The results of the dynamic range task with regard to both maximum P_s and intensity demonstrated no significant changes in these parameters during the training period. It is also thought that this task, in healthy subjects, did not utilize expiratory muscle activation to the extent that an increase in these parameters would be found during the training period. Certainly, louder speech may require expiratory muscle activation if the speaking task or singing phrase is held beyond the point that recoil forces are adequate for the P_s demand of the task. However, the dynamic range task utilized in this study did not require the participants to sustain the /pa/ syllable for a period beyond one or two seconds. Even if these subjects were able to generate much larger P_s, the mechanical properties of vocal fold vibration would limit excessively high rates of airflow at a certain point. Significant differences between baseline maximum P_s and maximum P_s during the detraining period were found for Group I. Likewise, a significant difference between baseline peak RMS and peak RMS during the detraining period was found for Group II. These significant effects indicate that these speech parameters continued to increase with continued visits with the investigator. Perhaps the participants increased their level of comfort with the task in the testing environment over time. Sex differences were noted in Group I for maximum Ps, but not for Group II. It is unknown why this occurred.

While increased expiratory muscle strength may not provide the ability to increase loudness in healthy subjects, it may serve to reduce excessive adductory activity during loud speech production. Generally, intensity and duration are maintained with increased medial compression of the vocal folds. Excessive compression of the vocal folds (hyperadduction) can lead to irritation of the laryngeal mucosa. It is hypothesized that having increased expiratory muscle strength, which allows for the ability to sustain the necessary P_s for a longer period of time, will reduce the need for medial compression, potentially reducing irritation and contact lesions in individuals with high speaking demands. More strenuous, longer speaking tasks at loud intensity levels, which require more sustained activation of the expiratory muscles may be necessary for methodological examination of speech alterations with training. Additionally, individuals with expiratory muscle weakness at the initiation of a program may demonstrate changes in these parameters. In fact, one of the major findings of a study previously mentioned (Cerny et al., 1997) indicates an increase in intensity and P_s in children with general muscle weakness.

It may also be of interest to measure physiologic changes in speech and voice parameters during or following activity that fatigues the expiratory muscles. It was previously discussed that the expiratory muscles play a role in torso stabilization, therefore, measuring parameters during or immediately following exercise that provides additional fatiguing effects to these muscles may reveal whether EMST is useful in overcoming this fatigue and improving speech production during strenuous activity levels (e.g. choreography). The use of expiratory muscle training as a compensatory strategy

for persons who do speak while engaged in high levels of activity has applications for musical theatre performers, aerobic instructors, and cheerleaders.

With regard to the detraining period, the P_s/MEP ratio remained significantly above baseline at the four-week data point. The P_0/MEP ratio remained significantly above baseline at both the four and eight week detraining data points. The findings reflect that very little expiratory muscle strength (MEP) was lost during the detraining period an important finding since information about detraining rates in respiratory muscles is lacking Likewise, information about detraining rates following other types of speech therapy programs has been poorly addressed. For example, if a therapy program is focused on strengthening the muscles involved in articulation and voice production, what is the impact of these strategies on improving the force of these muscles and if improvement is accomplished, how long does the effect last when the therapy is discontinued?

Ramig and colleagues (2001) provide results from one study that examined detraining of a therapy program in which the outcomes of the Lee Silverman Voice Treatment (LSVT) on improving speech parameters in patients with Parkinson's disease were followed two years after the treatment ended. This treatment program is similar to EMST in that it is a high effort, intensive program. Following the initial treatment program, participants had a 24% increase in vocal intensity (SPL) and a 32% increase in the standard deviation for semitones (STSD) during reading which was a measure used to indicate the amount of inflection in the voice. Two years after training, the participants lost approximately 8% of vocal intensity gained during treatment program. Additionally, participants lost approximately 4% of the STSD. These alterations in speech parameters

after two years were not statistically different from the data obtained after the four-week treatment program. In the current study, MEP was increased which led to a decrease in the P_s/MEP and P_0/MEP ratios for both groups and may lead to changes in vocal intensity and duration in individuals with expiratory muscle weakness. After an eight-week detraining period, the participants in both Group I and II only lost an average of 9% of the MEP gained during the training period (after an increase of approximately 50% from baseline during the training period). This loss was not statistically significant and minimal losses such as this may serve as a motivator for patients undergoing this type of treatment. It is proposed that future studies of EMST and its effects on speech production follow participants for longer durations (more than eight weeks) to determine when significant training effects begin to decline. It should be noted that detraining in EMST occurs when the training stimulus is completely removed. However, in LSVT, it is expected that the patient will continue to use the speaking patterns learned in therapy everyday after the treatment program is completed, thus providing a continuous training stimulus. It would be of interest to compare the results of a longer detraining period in EMST and LSVT.

A limitation of this training program is the pressure threshold limit on the trainer. Unfortunately, while this limit may reduce the ability to study individuals that initiate the program with high expiratory muscle strength, the pressure threshold cannot be raised due to safety concerns of generating pressures over 150 cmH_20 repeatedly because of the risk of stroke.

The benefits of the training program described in this study are that it produces relatively fast results. The training program is easy to learn and training sessions at home

last less than 15 minutes. These aspects of the program may lend to greater patient motivation and compliance compared to other therapy techniques.

Future directions in this line of research include the development of more sensitive measurement protocols to detect a change in voice and speech production following training. Particular attention will be directed toward speech tasks requiring greater, sustained P_s demands and monitoring alterations in perceived effort. Potential reductions in hyperfunction will be examined with these tasks as well using visual examination of the larynx. Application of EMST to clinical populations is currently being performed with patients with multiple sclerosis and Parkinson's disease to better understand how increasing strength can improve speech production in persons with skeletal muscle weakness and dysfunction.

Tables

Table 2-1. Spirometry values for all participants.

	Mean	SD
FEV_1 (L/s)		
Females	3.050	.399
Males	4.095	1.055
Average	3.442	.870
FVC (L/s)		
Females	3.526	.443
Males	4.789	1.054
Average	4.000	.957

Note. FEV_1 = forced expiratory volume in one second; FVC = forced vital capacity.

Table 2-2. Results of the multivariate repeated measures MANOVA examining the effect of weeks of training on all dependent variables

Training Group	Source	df	F	*p*
Group I	Week	24	5.184	.000*
Group II	Week	40	4.790	.000*

Note. *p<.05.

Table 2-3. Results of univariate analyses examining each dependent variable for Groups I and II.

Training Group	Source	Type III Sum of Squares	df[a]	Mean Square	F	p
Group I						
	MEP	19614.679	4.059	4832.738	17.058	.000*
	P_s/MEP	.004	2.841	.002	3.911	.017*
	P_0/MEP	.003	3.018	.001	6.112	.001*
	Maximum P_s	179.040	4.106	43.606	2.940	.027*
Group II						
	MEP	29322.751	4.609	6361.559	12.975	.000*
	P_s /MEP	.002	8.119	.001	1.483	.170
	P_0/MEP	.002	5.004	.001	3.211	.004*
	Maximum P_s	253.13	7.285	34.746	1.666	.123

Note. MEP = maximum expiratory pressure; P_s = subglottal pressure; P_0 = intraoral pressure.

[a]degrees of freedom were adjusted using the Huynh-Feldt correction factor as the sphericity assumption was not met.

*p<.05.

Table 2-4. Findings for the between-subject factor of sex for all dependent variables in Group I and II.

Source		df	Mean Square	F	p
Group I					
	MEP	1	14742.248	19.820	.001*
	P_s/MEP	1	.001	1.366	.262
	P_0/MEP	1	.001	2.242	.156
	Maximum P_s	1	231.136	8.509	.011*
Group II					
	MEP	1	6302.222	5.468	.035*
	P_s/MEP	1	.001	3.022	.104
	P_0/MEP	1	.001	3.357	.088
	Maximum P_s	1	218.635	2.516	.135

Note. MEP = maximum expiratory pressure; P_s = subglottal pressure; P_0 = intraoral pressure.

*p<.05.

Table 2-5. Maximum expiratory pressure (MEP) for all participants in Group I.

	Mean (cmH_20)	SD	Mean % above baseline
Baseline MEP			
Females	83.343	19.402	---
Males	140.600	29.174	---
Average	104.814	36.457	---
MEP after four weeks of training			
Females	123.353	24.527	48.00
Males	187.543	35.811	33.38
Average	147.424	42.644	40.65
Four weeks post-training			
Females	119.057	20.708	42.85
Males	177.150	46.881	25.99
Average	140.842	42.821	34.37
Eight weeks post-training			
Females	114.118	20.594	36.93
Males	173.810	37.580	23.62
Average	136.503	40.200	30.23

Table 2-6. Maximum expiratory pressure (MEP) for all participants in Group II.

	Mean (cmH_20)	SD	Mean % above baseline
Baseline MEP			
Females	78.794	19.794	---
Males	132.845	26.687	---
Average	99.063	34.682	---
MEP after eight weeks of training			
Females	132.174	39.021	67.74
Males	175.072	45.858	31.79
Average	148.261	45.547	49.66
Four weeks post-training			
Females	125.023	34.982	58.67
Males	164.256	50.031	26.38
Average	139.735	44.197	41.05
Eight weeks post-training			
Female	118.908	40.889	50.91
Male	167.898	48.394	26.39
Average	135.238	48.118	36.52

Table 2-7. Planned simple contrasts for maximum expiratory pressure (cmH_20) by week.

Group	Source	df	Mean Square	F	*p*
I					
	Baseline vs. wk4	1	28353.308	43.653	.000*
	Baseline vs. D1	1	19582.821	22.761	.000*
	Baseline vs. D2	1	15352.801	22.761	.000*
II					
	Baseline vs. wk8	1	34277.380	31.601	.000*
	Baseline vs. D1	1	22604.304	18.776	.001*
	Baseline vs. D2	1	19437.562	14.579	.002*

Note. wk4 = fourth and final week of training; wk8 = eighth and final week of training; D1 = fourth week of detraining period; D2 = eighth and final week of detraining period. *p<.05.

Table 2-8. Results of the subglottal pressure to maximum expiratory pressure ratio (P_s/MEP) for Group I.

	Mean	SD	% change from baseline
Baseline P_s/MEP			
Females	.068	.027	---
Males	.059	.029	---
Average	.065	.027	---
P_s/MEP after four weeks of training			
Females	.049	.011	-27.94
Males	.040	.018	-32.20
Average	.046	.014	-29.23
Four weeks post-training			
Females	.052	.008	-23.53
Males	.044	.023	-25.42
Average	.049	.015	-24.62
Eight weeks post-training			
Females	.052	.010	-23.53
Males	.049	.028	-16.95
Average	.053	.018	-18.46

Table 2-9. Results of subglottal pressure to maximum expiratory pressure ratio (P_s/MEP) for Group II.

	Mean	SD	% change from baseline
Baseline P_s/MEP			
Females	.079	.026	---
Males	.042	.012	---
Average	.064	.028	---
P_s/MEP after eight weeks of training			
Females	.055	.023	-30.38
Males	.044	.023	4.76
Average	.051	.023	-20.31
Four weeks post-training			
Females	.059	.022	-25.32
Males	.049	.021	16.66
Average	.055	.022	-14.06
Eight weeks post-training			
Females	.066	.032	-16.45
Males	.041	.013	- 2.38
Average	.058	.029	- 9.37

Table 2-10. Results of planned simple contrasts for the subglottal pressure to maximum expiratory pressure ratio (P_s/MEP) by week for Group I.

Source	df	Mean Square	F	*p*
Baseline vs. wk4	1	.005	8.386	.012*
Baseline vs. D1	1	.004	6.464	.023*
Baseline vs. D2	1	.002	2.689	.123

Note. wk4 = fourth and final week of training; D1 = fourth week of detraining period; D2 = eighth and final week of detraining period.
*p<.05.

Table 2-11. Results of the intraoral pressure to maximum expiratory pressure ratio (P_0/MEP) for Group I.

	Mean	SD	% change from baseline
Baseline P_0/Mep			
Females	.062	.027	---
Males	.050	.029	---
Average	.056	.027	---
P_0/MEP after four weeks of training			
Females	.044	.010	-29.03
Males	.032	.017	-36.00
Average	.040	.014	-28.57
Four weeks post-training			
Females	.044	.012	-29.03
Males	.035	.020	-30.00
Average	.041	.015	-26.78
Eight weeks post-training			
Females	.045	.009	-27.42
Males	.034	.016	-32.00
Average	.041	.013	-27.78

Table 2-12. Results of intraoral pressure to maximum expiratory pressure ratio (P_0/MEP) for Group II.

	Mean	SD	% change from baseline
Baseline P_0/Mep			
Females	.070	.025	---
Males	.034	.014	---
Average	.055	.028	---
P_0/MEP after eight weeks of training			
Females	.039	.015	-44.29
Males	.032	.020	-5.88
Average	.037	.016	-32.72
Four weeks post-training			
Females	.047	.014	-32.85
Males	.036	.017	5.88
Average	.043	.016	-21.81
Eight weeks post-training			
Females	.051	.020	-27.14
Males	.032	.015	- 5.88
Average	.045	.020	-18.81

Table 2-13. Planned simple contrasts for intraoral pressure to maximum expiratory pressure ratio (P_0/MEP) by week.

Group	Source	df	Mean Square	F	p
I					
	Baseline vs. wk4	1	.004	9.934	.007*
	Baseline vs. D1	1	.004	8.987	.010*
	Baseline vs. D2	1	.004	8.142	.013*
II					
	Baseline vs. wk8	1	.003	15.658	.001*
	Baseline vs. D1	1	.001	8.552	.011*
	Baseline vs. D2	1	.001	5.027	.042*

Note. wk4 = week 4 of training; wk8 = week 8 of training; D1 = four weeks post-training; D2 = eight weeks post-training.
*$p<.05$.

Table 2-14. Results of maximum subglottal pressure (P_s) for Group I.

	Mean (cmH_2O)	SD	% change from baseline
Baseline Maximum P_s			
Females	13.847	4.670	---
Males	20.522	11.201	---
Average	16.350	8.127	---
Maximum P_s after four weeks of training			
Females	14.387	4.130	3.89
Males	22.993	6.857	12.04
Average	17.614	6.665	7.73
Four weeks post-training			
Females	14.007	3.562	1.15
Males	23.100	6.255	12.56
Average	17.417	6.428	6.52
Eight weeks post-training			
Females	13.516	3.368	2.39
Males	27.957	6.439	36.23
Average	18.931	8.530	15.78

Table 2-15. Results of maximum subglottal pressure (P_s) for Group II.

	Mean (cmH_20)	SD	% change from baseline
Baseline Maximum P_s			
Females	14.406	6.649	---
Males	21.185	7.769	---
Average	17.117	7.657	---
Maximum P_s after eight weeks of training			
Females	16.003	6.349	11.08
Males	25.062	6.662	18.30
Average	19.022	7.624	11.13
Four weeks post-training			
Females	17.040	9.828	18.28
Males	25.520	11.732	20.46
Average	20.220	11.037	18.13
Eight weeks post-training			
Females	16.897	8.592	17.29
Males	24.392	12.039	15.14
Average	19.395	10.114	13.31

Table 2-16. Results of planned simple contrasts for maximum subglottal pressure (P_s) by week for Group I.

Source	df	Mean Square	F	*p*
Baseline vs. wk4	1	34.013	2.069	.172
Baseline vs. D1	1	28.119	.739	.405
Baseline vs. D2	1	189.251	6.072	.027*

Note. wk4 = fourth and final week of training; D1 = fourth week of detraining period; D2 = eighth and final week of detraining period.
*p<.05.

Table 2-17. Results of peak root mean square (RMS) for Group I.

	Mean (dB SPL)	SD	% change from baseline
Baseline peak RMS			
Females	101.678	3.684	---
Males	103.370	8.518	---
Average	102.242	5.489	---
Peak RMS after four weeks of training			
Females	102.526	4.961	.83
Males	108.102	7.786	4.58
Average	104.617	6.538	2.23
Four weeks post-training			
Females	98.624	3.648	-3.00
Males	104.748	4.045	1.33
Average	100.920	4.776	-1.29
Eight weeks post-training			
Females	100.620	4.956	-1.04
Males	108.793	4.776	5.24
Average	103.889	6.274	1.61

Table 2-18. Results of peak root mean square (RMS) for Group II.

	Mean (dB SPL)	SD	% change from baseline
Baseline peak RMS			
Females	105.467	10.716	---
Males	105.617	6.960	---
Average	105.526	9.106	---
Peak RMS after eight weeks of training			
Females	98.613	3.933	-6.49
Males	109.938	7.428	4.09
Average	102.860	7.729	-2.53
Four weeks post-training			
Females	98.829	6.155	-6.29
Males	103.930	6.468	-1.59
Average	100.742	6.571	-4.53
Eight weeks post-training			
Females	99.032	4.149	-6.10
Males	97.168	11.765	-7.99
Average	98.411	7.172	-6.74

Table 2-19. Results of planned simple contrasts for peak root mean square (RMS) by week for Group II.

Source	df	Mean Square	F	p
Baseline vs. wk8	1	25.366	.391	.542
Baseline vs. D1	1	264.170	7.995	.013*
Baseline vs. D2	1	847.478	10.802	.005*

Note. wk8 = eighth and final week of training; D1 = fourth week of detraining period; D2 = eighth and final week of detraining period.
*p<.05.

Table 2-20. Results for male participants in Group I who were able to train at 75% of their maximum expiratory pressure.

	MEP (cmH_20)	P_s/MEP	P_0/MEP	Maximum P_s (cmH_20)	Peak RMS (dB SPL)
Baseline					
Mean	133.500	.068	.055	16.680	100.286
SD	34.567	.027	.041	2.986	10.040
% change from baseline	---	---	---	---	---
After four weeks of training					
Mean	159.510	.049	.037	20.507	103.053
SD	25.952	.023	.025	4.730	6.690
% change from baseline	19.48	- 27.94	-32.72	22.94	2.76
After four weeks of detraining					
Mean	136.700	.060	.047	21.826	101.887
SD	23.995	.023	.029	5.493	2.718
% change from baseline	2.39	-11.76	-14.55	30.85	1.59
After eight weeks of detraining					
Mean	141.467	.070	.044	27.740	106.730
SD	19.807	.029	.019	4.409	4.921
% change from baseline	5.96	2.94	-20.00	66.31	6.42

Note. MEP = maximum expiratory pressure; P_s = subglottal pressure; P_0 = intraoral pressure; RMS = root mean square.

Table 2-21. Results for male participants in Group II who were able to train at 75% of their MEP.

	MEP (cmH_20)	P_s/MEP	P_0/MEP	Maximum P_s (cmH_20)	Peak RMS (dB SPL)
Baseline					
Mean	126.393	.046	.034	21.018	103.560
SD	29.876	.012	.014	9.534	8.429
% change from baseline	---	---	---	---	---
After eight weeks of training					
Mean	154.915	.056	.037	26.523	111.807
SD	42.966	.023	.027	3.763	8.251
% change from baseline	25.56	21.73	8.82	26.19	7.96
After four weeks of detraining					
Mean	141.858	.060	.041	26.472	102.803
SD	46.167	.014	.020	13.318	7.227
% change from baseline	12.23	30.43	20.58	25.94	- .73
After eight weeks of detraining					
Mean	142.077	.050	.038	24.492	109.674
SD	44.488	.065	.020	13.39	2.367
% change from baseline	12.40	8.69	11.76	16.52	5.90

Note. MEP = maximum expiratory pressure; P_s = subglottal pressure; P_0 = intraoral pressure; RMS = root mean square.

Table 2-22. Inter-measurer reliability results and significance for all speech parameters

	r	*p*
P_s	.951	.114
P_0	.792	.537
Maximum P_s	.983	.381

Note. P_s = subglottal pressure; P_0 = intraoral pressure.

Table 2-23. Intra-measurer reliability results and significance for all speech parameters

	r	*p*
P_s	.999	.577
P_0	.967	.171
Maximum P_s	.992	.126

Note. P_s = subglottal pressure; P_0 = intraoral pressure.

Figures

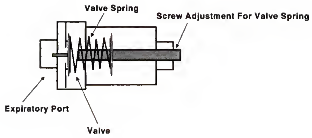

Figure 2-1. Schematic drawing of the expiratory pressure threshold trainer.

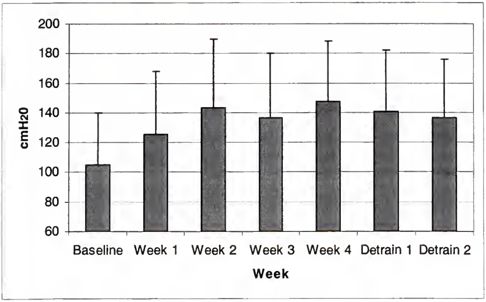

Figure 2-2. Mean maximum expiratory pressure across all training and detraining periods for Group I.

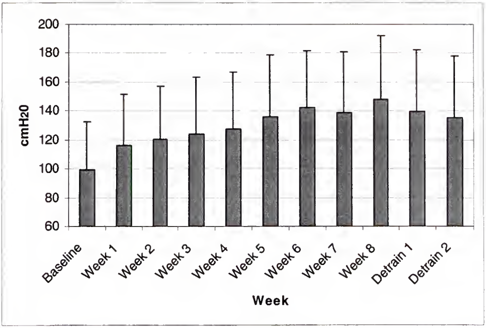

Figure 2-3. Mean maximum expiratory pressure across all training and detraining periods for Group II.

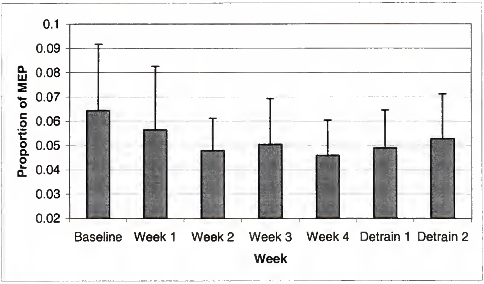

Figure 2-4. Mean of the subglottal pressure to maximum expiratory pressure ratio (P_s/MEP) across the training and detraining periods for Group I.

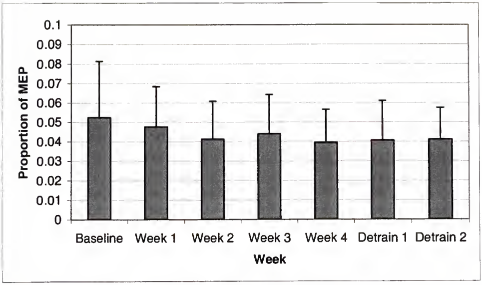

Figure 2-5. Mean of the intraoral pressure to maximum expiratory pressure ratio (P_0/MEP) across the training weeks and the detraining period for Group I.

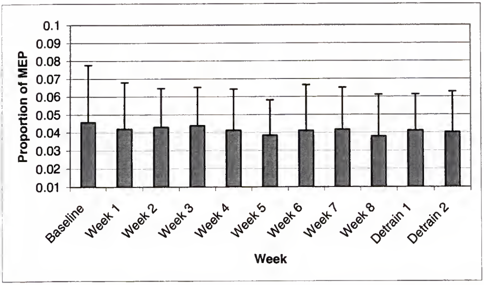

Figure 2-6. Mean of the intraoral pressure to maximum expiratory pressure ratio (P_0/MEP) across the training and detraining periods for Group II.

CHAPTER 3
COUGH PRODUCTION WITH INCREASED EXPIRATORY MUSCLE STRENGTH

Introduction

The cough reflex is a defense mechanism for clearance of the respiratory tract. This reflex is complex in that it relies on sensory receptors throughout the respiratory system which provide information to a central cough center which is thought to be located in the medulla (Bouros et al., 1995; Chou & Wang, 1975; Dawid-Milner et al., 1993). This center is also responsible for specific control of the muscles that execute cough production. Stimulation of the cough reflex usually occurs as a result of a foreign substance or excessive mucus in the airways. Cough production consists of three phrases: inspiration, glottal compression, and expiration (Shah & Shah, 2001). During inspiration, the glottis opens and allows air to fill the lungs. The glottis closes via adductory movement of the vocal folds and the expiratory muscles contract, building intrathoracic pressure. Finally, the glottis opens via contraction of the vocal abductor muscles and the air is expired from the lungs with great velocity.

Cough production may be compromised due to weakness of the inspiratory or expiratory muscles, structural changes in the airway, or excess mucus. Following a large inspiration, the abdominal and internal intercostal muscles rapidly contract to send an explosion of air through the glottis. The mean expiratory flow rate during cough is between 5 and 12 L/sec in healthy individuals (Arora & Gal, 1981; Langlands, 1967; Leiner et al., 1966). The high velocity of airflow through the lower airways generates

turbulence causing secretions to move above the level of the larynx. The inability to generate appropriate airflow velocity for clearance of the airway results in pulmonary complications, due to aspiration or an inability to clear excessive secretions (Holas et al., 1994; Horner et al., 1988).

Cough magnitude may also be compromised due to poor adductory movement of the vocal folds. A disruption to the innervation of the laryngeal muscles (i.e., thyroarytenoid and lateral cricoarytenoid) as the result of central or peripheral nervous system damage may result in incomplete adduction of the glottis. Incomplete closure of the glottis decreases the amount of intrathoracic pressure developed during the compression phase of the cough, thus potentially limiting the magnitude of airflow rates. Individuals with poor laryngeal function are at high risk for aspiration due to poor airway protection as well as reduced strength of cough (Addington et al., 1999; Smith-Hammond et al., 2001).

Significant decreases in inspiratory or expiratory muscle strength results in decreased expiratory peak flows that are necessary for effective cough (McCool & Leith, 1987). Reduction in the velocity of airflow during cough production has been correlated with poor expiratory muscle strength in individuals with neuromuscular disease including amyotrophic lateral sclerosis (ALS), muscular dystrophy (Polkey et al., 1998; Suarez et al., 2002; Szeinberg et al., 1988) and spinal cord injury (DiMarco et al., 1995; Taylor et al., 2002). Decreased respiratory muscle strength is one of the major causes of morbidity and eventual death in individuals with neuromuscular disease (Gosselink et al., 2000; Inkley et al., 1974). The inability to effectively clear the airway with an adequate cough may result in mucus plugs in the lower airway and/or aspiration of food and liquid. A

study of the relationship between expiratory muscle strength and airflow velocity in healthy participants was performed by inducing skeletal muscle weakness with d-turbocurarine. A slight decrease in maximum expiratory flow rate during a voluntary cough was noted with the induced muscle weakness. The effect was hypothesized to be due to both inspiratory and expiratory muscle weakness (Arora, & Gal, 1981).

Cough magnitude can be increased using several assistive means that work to increase expiratory driving force These techniques have been used primarily for individuals with spinal cord injury. Increases in peak expiratory flow rate occur when a caregiver manually assists coughing by providing an abdominal thrust maneuver with their hand. Increases in peak expiratory flow also occur with mechanical insufflation (Bach et al, 1993; Sivasothy et al., 2001) as well as electrical simulation of the abdominal muscles (Jaeger et al., 1993; Taylor et al., 2002).

The success with these techniques in increasing cough airflow rate lends credence to the hypothesis that increasing expiratory muscle strength would produce similar effects. Smeltzer and colleagues (1996) completed a study in which an expiratory pressure threshold training program was implemented with ten participants diagnosed with clinical definite multiple sclerosis (MS). The participants completed the training with an expiratory pressure threshold device for three months. The group mean increase in maximum expiratory muscle strength after training, measured as maximum expiratory pressure (MEP), was 37%. The participants reported that they experienced a decrease in choking while eating. The authors hypothesized that the subjective improvements in swallow function reported by these patients might be related to an improved ability to clear the airway with an adequate cough.

In another study, Gosselink and colleagues (2000) implemented a three-month expiratory pressure threshold training program with nine patients with MS. The group mean increase in MEP was approximately 25%. An interesting finding of this study was that the mean maximum inspiratory pressure (MIP) actually increased more significantly than MEP. The authors hypothesized that expiratory muscle training allowed the diaphragm to contract at a more advantageous position due to increases in expiratory strength. Subjective reports of improvement in cough effectiveness were noted following the training program. Reports of improved cough effectiveness were maintained up to three months following training.

While one study thus far provides subjective evidence of improved cough with expiratory muscle strength training (EMST), the purpose of the current study was to examine specific physiologic changes that occur in cough parameters following EMST. A methodological study with healthy participants was used to study these potential changes. Several hypotheses were made regarding changes in cough parameters. The first hypothesis was that maximum expiratory flow rate during voluntary cough would increase as expiratory muscle strength increased. The second hypothesis was that the time from when the glottis is open following the compression phase to the point of maximum flow rate (rise time) would decrease due to the increase in the airflow velocity generated by increased expiratory muscle strength. The third hypothesis was that the compression phase time would decrease due to increased input to subglottic pressure receptors that signal central cough centers located in the brainstem that sufficient pressure has been achieved for appropriate airflow rates. Participants in this study were enrolled in either a four-week or eight-week training program. It was hypothesized that greater

strength gains would be achieved with the longer training program and thus lead to greater alterations to the cough parameters of interest.

Methods

Thirty-two healthy participants completed this study. Twelve participants were males. The age of the males ranged from 18 to 32 years with an average age of 24.6 years. Twenty participants were females. The age of the females ranged from 19 to 48 years with an average age of 25.6 years Participants were recruited from the Gainesville, Florida, area.

All participants' MEP values were within a normative range for age and sex (Black & Hyatt, 1969). Pulmonary function testing was performed as a screening measure. All participants had forced expiratory volumes in the first second (FEV1) and forced vital capacity (FVC) within normal limits (American Thoracic Society, 1987; see Table 3-1). All participants had a negative history of chronic and acute cardiac disease, upper respiratory infection, pulmonary dysfunction/disease, neuromuscular disease, immune system disease, vocal disturbances (chronic or acute), obesity, and no history of smoking within last five years (tobacco or recreational drugs).

Only those participants who were able to maintain their current level of physical activity (including both aerobic exercise and weightlifting) throughout the entire training period were recruited. Participants were asked to specifically report any significant changes in their level of activity throughout their participation in the study with regards to intensity and frequency of exercise. An example of a significant change was defined as a sedentary person who begins exercising two to four days per week. Participants were discontinued in the study if they made a significant change in the activity level as

described above. Extreme athletes such as marathon runners and competitive weightlifters were also excluded.

Dependent Measures

Maximum expiratory pressure

Expiratory muscle strength was measured indirectly as the maximum expiratory pressure (MEP) at the mouth. The measurement apparatus consisted of a mouthpiece connected to a pressure manometer by 50 cm of 2 mm i.d. tubing with a 14-guage-needle air-leak. In order to measure MEP, the participants' nose was occluded with nose clips. After inhaling to total lung capacity, the participant placed his or her lips around the mouthpiece and blew out as forcefully as possible. Three repeated measures were taken with a one-minute rest between trials, until the measurements obtained were within 5% of each other. The average of these three values was used.

Cough parameters

The expiratory flow waveform during a voluntary cough was obtained using a mouthpiece attached directly to a non-heated pneumotachograph (Hans Rudolf) with 30 cm of plastic tubing (3 mm diameter). The pneumotachograph was attached to a differential pressure transducer (ML 140) with a pressure sensing range of +/- 12.5 cmH_20. This attachment was fitted directly into the Power Lab/4SP data acquisition system (ADInstruments, ML750). Hardware was provided by ADInstruments for calculation of spirometric measures. The pneumotachograph was calibrated by injecting a volume of three liters of air using a calibrated syringe. Volume and flow were calculated using a calibration routine within Chart 4 for Windows software (ADInstruments). All cough signals were also recorded using Chart 4 software. The participants were asked to

produce a "strong" voluntary cough into the pneumotachograph. The procedure was completed three times with a 30 second break between coughs. All cough parameters were measured using the Chart 4 software. A second investigator measured ten percent of the dataset to determine intermeasurer reliability.

The following parameters of cough were obtained from the flow waveform (Figure 3-1): maximum flow rate (MF), compression time (CT), and rise time (RT). Maximum expiratory flow (L/s) during cough was defined as the peak flow rate measured from the expiratory flow waveform produced during the voluntary coughs. Compression time (in seconds), defined as the time at the end of the inspiratory phase to the beginning of the expiratory phase, was measured from the recorded cough signals. Rise time (in seconds), defined as the time the compression phase ends to the highest peak of flow during the cough, was measured from the recorded samples.

Training Procedures

Expiratory pressure threshold trainer

An expiratory pressure threshold trainer was used to complete the EMST program. This cylindrical device consists of a mouthpiece and a one-way spring-loaded valve (Figure 3-2). The valve blocks expiratory airflow until a sufficient threshold pressure is reached to overcome the spring force. To achieve this threshold pressure, the participant breathed out with increased expiratory effort. As long as the threshold pressure is maintained, air flows through the device. The device contains an adjustable spring, which allowed the required threshold pressure to be increased.

Training protocol

Each participant was assigned to one of two training groups: a four-week training group or an eight-week training group. Each of the groups will be referred to as Group I or Group II, respectively. Two training lengths were utilized in this study to examine the effect of greater training durations on cough parameters. The participants in each of the two groups were matched for sex due to expected differences in male and female strength gains. Males typically demonstrate greater increases in skeletal muscle strength in response to strength training programs (Ivey et al., 2000; Lewis et al., 1986). Likewise, greater increases in MEP were noted in males compared to females in a previously described study that utilized pressure-threshold training in healthy participants (Sapienza et al., 2002).

Group I. These participants completed a pressure threshold expiratory training program for four weeks. The participants performed expiratory breathing exercises with the pressure threshold trainer five days per week. The training session consisted of five sets of five breaths at a threshold set at 75% of the participants' MEP. The participant completed the training sessions throughout the week independently at home. The pressure threshold was increased each week of training to reflect 75% of their MEP.

Group II. These participants completed the same program described for Group I, however, they trained for a total of eight weeks.

Detraining. Both groups were followed for eight weeks after the completion of the training program to further examine the relationship between expiratory muscle strength and the cough parameters. All of the dependent measures (maximum expiratory

flow, compression time, rise time) were obtained again at four-weeks post-training and eight-weeks post-training.

Compliance

Participant compliance was acknowledged through participant education on the use of the device, mid-week contact by the investigator, and completion of a training log. Participants were provided with written and verbal instructions for the use of the device.

Statistical Analyses

The primary statistical method that was used to examine treatment differences with respect to the change from baseline scores across the treatment groups for MEP was a multivariate repeated measures analysis of variance (MANOVA). A separate MANOVA was performed for Group I and Group II because of the difference in the number of within-subject factor levels (four-weeks vs. eight-weeks). Week was the within-subject factor and sex was the between-subject factor. Missing values in the data set were replaced using a linear trend function for the predicted values. There were 11 missing values in the entire data set which is less than 1%. Significant differences at alpha'=0.05 were tested using univariate comparisons. Planned simple contrasts were utilized to examine differences between dependent variables to the baseline measures across the weeks of training.

Results

A significant main effect was found for the within-subject factor of week for both of the MANOVAs performed for Group I and II (Table 3-2). Univariate tests for each of the dependent variables (Table 3-3) revealed a significant effect for MEP for both Group

I and II. The only cough parameter that revealed a significant effect was compression time in Group II.

A main effect for the between-subject factor of sex was found for both Group I $F(4,15) = 11.008$, $p =< .05$. and Group II $F(4, 15) = 3.903$, $p <.05$. Univariate tests for each of the dependent variables revealed a significant difference between males and females for MEP and MF in both groups (Table 3-4).

Increase in MEP

Means and standard deviations as well as percent increase from baseline for MEP across the training and detraining period are in Tables 3-5 and 3-6. Figure 3-2 and 3-3 show the change in MEP over the training and detraining periods. Given the significant univariate results for MEP, analyzing the simple contrasts further helped interpret the effect. Contrasts showed that the baseline MEP was significantly different at the final week of training for both Groups I and II. As well, significant differences between baseline MEP and MEP at the end of the fourth week and eighth week of the detraining period were found for both training groups. (Table 3-7).

Cough Parameters

Means and standard deviations as well as percent increase from baseline for maximum flow rate (MF), compression time (CT), rise time (RT) across the training and detraining period are presented in Tables 3-8 through 3-13. As the univariate analysis revealed a significant result for CT in Group II, planned simple contrast were performed. Contrasts showed that baseline CT was not significantly different at the eighth week of training. However, baseline CT and CT at the end of the fourth week and eighth week of the detraining period were found to be significant (Table 3-14).

Trainer Limitations

Six of the male participants (three in Group I; three in Group II) achieved a MEP above 200 cmH_20 before the completion of the training program. The trainer was set to reflect 75% of the participants' MEP obtained each week. The maximum pressure threshold setting on the trainer used for this study was 150 cmH_20. Those participants who achieved an average MEP above 200 cmH_20 during the training program continued to train with the trainer set at 150 cmH_20, however, this was at a pressure threshold less than 75% of their MEP. So even though these participants had achieved an average MEP greater than 200 cmH_20, the training thresholds were unable to be raised in order to protect the participant from any potential risks that could have occurred when generating expiratory pressures above 150 cmH_20 repeatedly. Tables 3-15 and 3-16 present data from only those male participants who were able to train at 75% of their MEP throughout the entire training program.

Reliability

Pearson r correlations were used to determine the reliability between the measurers as well as t-tests to test for differences between the data sets. The Pearson r correlation results are in Table 3-17. Strong correlations were noted with all data sets and no significant differences in the data set were found according to the t-tests ($p = 0.05$).

Additionally, the primary investigator measured approximately 10% of all the dependent variables again. Pearson r correlations were used to determine the reliability as well as t-tests for differences between the measures. The Pearson r correlation results are in Table 3-18. Strong correlations were found will all data sets and no significant differences were found between the data sets according to the t-tests ($p < 0.05$).

Discussion

The results of this study revealed a significant increase in MEP in both Group I and II. The average increase from baseline across both groups was 45%. Similar increases have been found in other studies using expiratory pressure threshold training with positive increases in MEP occurring at about four weeks of training. The current study further supports that EMST is effective in increasing expiratory muscle strength. It also suggests that EMST has the potential to impact cough production parameters due to the increased pressure generating capability of the expiratory muscles.

Maximum expiratory flow and rise time did not change significantly with expiratory muscle training in these participants. Both of these measures are flow-dependent and flow limitations occur during forced expiration due to dynamic airway compression which prevents airway collapse (West, 2000). All of the participants in this study were able to generate maximum flow rates during cough within the normal range at baseline. Therefore, additional expiratory force would potentially only increase compression of the airways offering increased resistance to the flow. Individuals with expiratory muscle weakness and whose initial maximum flow rate values do not exceed flow rate limitations of the airways may have different results. These individuals will likely increase their maximum flow rate with EMST because they are not initially capable of generating high forces on the lungs. With EMST, these individuals will increase their force generating capability and thus have an increased flow rate during cough. Increased flow rates have been found in individuals with spinal cord injury with techniques that increase expiratory force (i.e., manually assisted cough and mechanical insufflation). Expiratory muscle strength training has the potential to provide a similar

effect as a manually assisted cough without the need for another person to provide the increased expiratory force.

The finding that compression time decreased is perhaps due to the increase in the force output of the expiratory muscles. While compression time did not decrease overall for Group I, it did decrease by 58% in Group II. The decrease in compression time from baseline to the end of training was near significant and the change at the fourth and eighth week of detraining compared to baseline were statistically significant. Close inspection of the individual participants revealed that 23 out of 32 participants had a declining trend in compression time. Reasons for the decrease in compression time may be due to the presence of airway receptors and their function. A variety of airway receptors are responsible for controlling the cough reflex. Unfortunately, at this time, the neural control of cough in not completely understood (Widdicombe, 1995). However, several researchers indicate that a central cough center exists in the medulla near the nucleus solitarius (Bouros et al., 1995; Chou & Wang, 1975; Dawid-Milner et al., 1993). Mechanoreceptors located in the subglottal area are responsible for detecting changes in pressure during breathing and cough production. Information regarding changes in pressure is sent back to the control center via sensory branches of the vagus nerve (Bouros et al., 1995; Sant'Ambrogio et al., 1983; Widdicombe, 1982; 1995; 2001). It is hypothesized that increased expiratory muscle strength which results in potentially greater pressure generating capability is detected by these receptors as faster changes in pressure in the subglottal area. As a result, alterations may occur in the medulla cough control center that inhibit efferent outputs to the adductory laryngeal muscles, thus decreasing glottal compression time.

Certainly, the control of motor efferents for cough is not viewed as exclusive to the medulla for the production of voluntary cough. Cortical and subcortical areas are known to influence the cough center particularly for cough suppression, but are also necessary for the initiation of a voluntary cough. Alterations in compression time with increased expiratory muscle strength would be of future interest in an induced reflexive cough to study this interaction without the influence of cortical involvement.

Reducing compression time with EMST has implications for those with adductory vocal fold muscle weakness whereby compression of the vocal folds cannot be achieved in the same manner as healthy individuals. For example, decreasing the necessary compression time for persons with laryngeal weakness caused by age related decreases in vocal fold muscle function or neurological impairment, may improve cough efficiency. Older individuals tend to be more at risk for swallowing dysfunction due to weakness of the bulbar musculature. As stated previously, this weakness increases the risk of aspiration. Increasing expiratory force generation may be a compensatory mechanism to assist cough production when adductory force of the vocal folds is compromised.

Further investigation of the correlation between expiratory muscle strength and cough production is warranted due to its potential implications for improvement of cough function in individuals with upper airway weakness or dysfunction. Current study of cough parameters and how they may be affected with EMST is being conducted in individuals with multiple sclerosis, Parkinson's disease, and spinal cord injury.

Tables

Table 3-1. Spirometry values for all participants.

	Mean	SD
FEV_1 (L/s)		
Females	3.050	.399
Males	4.095	1.055
Average	3.442	.870
FVC (L/s)		
Females	3.527	.473
Males	4.789	1.054
Average	4.000	.957

Note. FEV_1 = forced expiratory volume in one second; FVC = forced vital capacity.

Table 3-2. Results of the multivariate repeated measures MANOVA examining the effect of weeks of training on all dependent variables.

Training Group	Source	df	F	*p*
Group I	Week	24	5.028	.000*
Group II	Week	40	4.321	.000*

Note. $^*p<.05$.

Table 3-3. Results of univariate analyses examining each dependent variable for Groups I and II.

Training	Source	Type III Sum of Squares	df[a]	Mean Square	F	*p*
Group I						
	MEP	19614.679	4.059	4832.738	17.058	.000*
	MF	11.958	4.370	2.736	.698	.608
	CT	.306	4.198	.007	.795	.538
	RT	.005	4.418	.001	.841	.514
Group II						
	MEP	29322.751	4.609	6361.559	12.975	.000*
	MF	15.520	5.471	1.552	.951	.489
	CT	1.498	3.991	.375	2.588	.047*
	RT	.002	3.735	.006	1.414	.244

Note. MEP = maximum expiratory pressure; MF = maximum flow rate; CT = compression time; RT = rise time.
[a]degrees of freedom were adjusted using the Huynh-Feldt correction factor as the sphericity assumption was not met.
$^*p<.05$.

Table 3-4. Findings for the between subject factor of sex for all dependents variables in Group I and II.

Source		df	Mean Square	F	*p*
Group I					
	MEP	1	14742.248	19.820	.001*
	MF	1	23.924	13.506	.002*
	CT	1	.341	2.702	.122
	RT	1	.001	.143	.711
Group II					
	MEP	1	6302.222	5.468	.035*
	MF	1	42.502	17.132	.001*
	CT	1	.050	.794	.388
	RT	1	.004	3.783	.072

Note. MEP = maximum expiratory pressure; MF = maximum flow rate; CT = compression time; RT = rise time.
$*p<.05$.

Table 3-5. Maximum expiratory pressure (MEP) for all participants in Group I.

	Mean (cmH_20)	SD	Mean % above baseline
Baseline MEP			
Females	83.343	19.402	---
Males	140.600	29.174	---
Average	104.814	36.457	---
MEP after four weeks of training			
Females	123.353	24.527	48.00
Males	187.543	35.811	33.38
Average	147.424	42.644	40.65
Four weeks post-training			
Females	119.057	20.708	42.85
Males	177.150	46.881	25.99
Average	140.842	42.821	34.37
Eight weeks post-training			
Females	114.118	20.594	36.93
Males	173.810	37.580	23.62
Average	136.503	40.200	30.23

Table 3-6. Maximum expiratory pressure (MEP) for all participants in Group II.

	Mean (cmH_20)	SD	Mean % above baseline
Baseline MEP			
Females	78.794	19.794	---
Males	132.845	26.687	---
Average	99.063	34.682	---
MEP after eight weeks of training			
Females	132.174	39.021	67.74
Males	175.072	45.858	31.79
Average	148.261	45.547	49.66
Four weeks post-training			
Females	125.023	34.982	58.67
Males	164.256	50.031	26.38
Average	139.735	44.197	41.05
Eight weeks post-training			
Female	118.908	40.889	50.91
Male	167.898	48.394	26.39
Average	135.238	48.118	36.52

Table 3-7. Planned simple contrasts for maximum expiratory pressure (MEP) by week.

Group	Source	df	Mean Square	F	*p*
Group I					
	Baseline vs. wk4	1	28353.308	43.653	.000*
	Baseline vs. D1	1	19582.821	22.761	.000*
	Baseline vs. D2	1	15352.801	22.783	.000*
Group II					
	Baseline vs. wk8	1	34277.380	31.601	.000*
	Baseline vs. D1	1	22604.304	18.776	.001*
	Baseline vs. D2	1	19437.562	14.579	.002*

Note. wk4 = fourth and final week of training; wk8 = eighth and final week of training; D1 = fourth week of detraining period; D2 = eighth and final week of detraining period. *p<.05.

Table 3-8. Results of maximum flow rate (MF) for Group I.

	Mean (L/s)	SD	% change from baseline
Baseline MF			
Females	6.460	2.190	---
Males	10.685	3.290	---
Average	8.044	3.309	---
MF after four weeks of training			
Females	7.447	1.314	15.27
Males	8.963	1.190	-16.11
Average	8.015	1.443	-.36
Four weeks post-training			
Females	7.552	1.51	16.90
Males	10.217	3.018	-4.37
Average	8.551	2.486	6.30
Eight weeks post-training			
Females	7.263	1.893	12.43
Males	10.380	3.022	-2.85
Average	8.432	2.761	4.82

Table 3-9. Results of maximum flow rate (MF) for Group II.

	Mean (L/s)	SD	% change from baseline
Baseline MF			
Females	7.332	2.111	---
Males	10.415	1.899	---
Average	8.488	2.500	---
MF after eight weeks of training			
Females	7.836	1.436	6.87
Males	11.443	2.549	9.87
Average	9.189	2.580	8.26
Four weeks post-training			
Females	7.606	1.497	3.73
Males	11.474	2.436	10.16
Average	9.056	2.658	6.69
Eight weeks post-training			
Females	7.904	1.565	7.80
Males	11.077	2.446	6.35
Average	8.962	2.383	5.58

Table 3-10. Results of compression time (CT) for Group I.

	Mean (s)	SD	% change from baseline
Baseline CT			
Females	.480	.425	---
Males	.861	.585	---
Average	.623	.508	---
CT after four weeks of training			
Females	.358	.227	-25.41
Males	.852	.738	-1.04
Average	.543	.523	-12.84
Four weeks post-training			
Females	.330	.262	-31.25
Males	.729	.499	-15.33
Average	.479	.405	-23.11
Eight weeks post-training			
Females	.439	.428	- 8.54
Males	.533	.353	-38.10
Average	.474	.392	-23.92

Table 3-11. Results of compression time (CT) for Group II.

	Mean (s)	SD	% change from baseline
Baseline CT			
Females	.825	.618	---
Males	.410	.319	---
Average	.669	.553	---
CT after eight weeks of training			
Females	.344	.154	- 58.30
Males	.382	.463	- 6.83
Average	.358	.294	-46.48
Four weeks post-training			
Females	.316	.165	-61.69
Males	.272	.264	-33.65
Average	.299	.200	-55.31
Eight weeks post-training			
Females	.274	.088	-66.79
Males	.283	.218	-30.96
Average	.276	.137	-58.74

Table 3-12. Results of rise time (RT) for Group I.

	Mean (s)	SD	% change from baseline
Baseline RT			
Females	.112	.047	---
Males	.137	.096	---
Average	.122	.068	---
RT after four weeks of training			
Females	.118	.034	5.35
Males	.128	.126	-6.57
Average	.122	.077	0
Four weeks post-training			
Females	.112	.038	0
Males	.131	.109	-4.38
Average	.119	.070	-2.45
Eight weeks post-training			
Females	.120	.034	7.14
Males	.123	.087	-1.02
Average	.121	.056	-.82

Table 3-13. Results of rise time (RT) for Group II.

	Mean (s)	SD	% change from baseline
Baseline RT			
Females	.104	.035	---
Males	.060	.026	---
Average	.088	.038	---
RT after eight weeks of training			
Females	.100	.032	- 3.84
Males	.069	.019	15.00
Average	.089	.031	1.14
Four weeks post-training			
Females	.111	.066	6.73
Males	.089	.041	48.33
Average	.103	.057	17.05
Eight weeks post-training			
Females	.108	.036	3.85
Males	.064	.021	6.67
Average	.093	.037	5.68

Table 3-14. Planned simple contrasts for compression time (CT) by week.

Source	df	Mean Square	F	*p*
Baseline vs. wk8	1	.744	3.881	.069
Baseline vs. D1	1	.519	9.486	.008*
Baseline vs. D2	1	.682	6.433	.024*

Note. wk8 = eighth and final week of training; D1 = fourth week of detraining period; D2 = eighth and final week of detraining period.
*p<.05.

Table 3-15. Results for male participants in Group I who were able to train at 75% of their maximum expiratory pressure.

	MEP(cmH_20)	MF (L/s)	CT (s)	RT(s)
Baseline				
Mean	133.500	12.100	1.066	.080
SD	34.567	4.426	.744	.026
% change from baseline	---	---	---	---
After four weeks of training				
Mean	159.510	8.967	1.091	.084
SD	25.952	.179	.903	.051
% change from baseline	19.17	-25.89	2.34	5.00
After four weeks of detraining				
Mean	136.700	10.963	.914	.104
SD	23.995	4.280	.541	.094
% change from baseline	2.39	-9.39	-14.25	30.00
After eight weeks of detraining				
Mean	141.467	10.550	.709	.090
SD	19.807	3.579	.310	.057
% change from baseline	5.97	-12.81	-33.48	12.50

Note. MEP = maximum expiratory pressure; MF = maximum flow rate; CT = compression time; RT = rise time.

Table 3-16. Results for male participants in Group II who were able to train at 75% of their maximum expiratory pressure.

	MEP(cmH_20)	MF (L/s)	CT (s)	RT(s)
Baseline				
Mean	126.393	10.538	.434	.070
SD	29.877	2.287	.395	.027
% change from baseline	---	---	---	---
After eight weeks of training				
Mean	154.915	11.033	.439	.079
SD	42.966	2.860	.558	.015
% change from baseline	22.56	4.70	1.15	12.85
After four weeks of detraining				
Mean	141.858	10.800	.292	.089
SD	46.167	2.482	.299	.046
% change from baseline	12.24	2.49	-32.71	27.14
After eight weeks of detraining				
Mean	142.077	10.076	.313	.052
SD	44.489	2.381	.235	.013
% change from baseline	12.40	-4.38	-27.88	-25.71

Note. MEP = maximum expiratory pressure; MF = maximum flow rate; CT = compression time; RT = rise time.

Table 3-17. Inter-measurer reliability results and significance for all cough parameters.

	r	*p*
MF	.994	.303
CT	.982	.445
RT	.921	.391

Note. MF = maximum flow rate; CT = compression time; RT = rise time.

Table 3-18. Intra-measurer reliability results and significance for all cough parameters.

	r	*p*
MF	.993	.996
CT	.782	.300
RT	.969	.154

Note. MF = maximum flow rate; CT = compression time; RT = rise time.

Figures

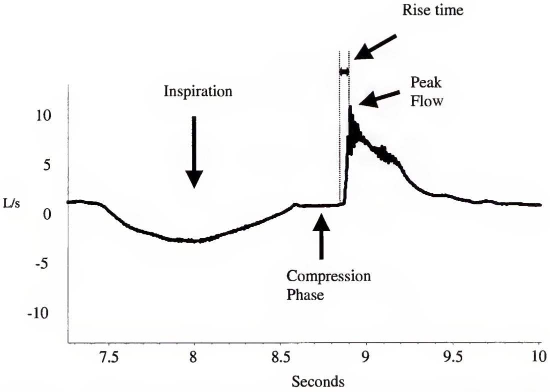

Figure 3-1. Measurements taken from airflow waveform during a voluntary cough.

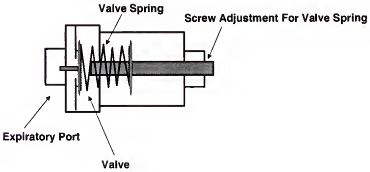

Figure 3-2. Schematic drawing of the expiratory pressure threshold trainer.

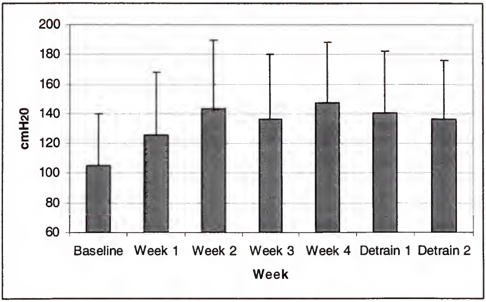

Figure 3-2. Mean maximum expiratory pressure across all training and detraining periods for Group I.

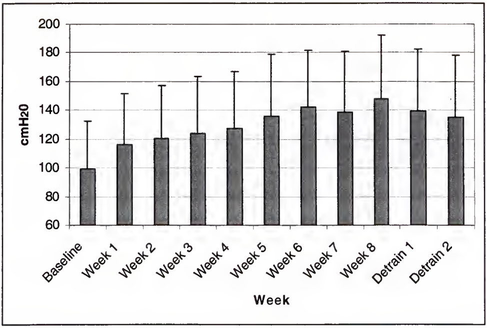

Figure 3-3. Mean maximum expiratory pressure across all training and detraining periods for Group II.

CHAPTER 4
STRENGTH TRAINING IN EXPIRATORY MUSCLES

Introduction

The current literature suggests that expiratory muscle strength training (EMST) programs are capable of increasing expiratory muscle strength (Cerny et al., 1997; Gosselink et al., 2000; Hoffman Ruddy et al., 2001; O'Kroy & Coast, 1993; Sapienza et al., 2002; Smeltzer et al., 1996; Suzuki et al., 1995). It has been hypothesized that increasing expiratory muscle strength may be useful in improving speech and cough production. The majority of the studies utilizing EMST thus far have been relatively short in length (approximately four weeks). These short training durations limit the understanding of the response of the expiratory muscles to longer training durations and do not allow strength patterns to be characterized. For example, it is unknown when plateaus in strength begin and/or when maximal training effects are achieved.

As skeletal muscles, both the respiratory and limb muscles are structurally and functionally similar (Powers et al., 1997). These similarities are known from muscle biopsies. Respiratory muscle biopsies reveal that abdominal muscles (primary expiratory muscles) are composed of an approximately equal distribution of type I (54%) and type II (46%) muscles fibers. The internal intercostal muscles, however, consist of slightly fewer type II fibers (62%) (Mizuno, 1991). Most limb muscle has an equal distribution of type I and type II fibers (Powers & Howley, 2001). Type I or slow-twitch fibers assist more often during endurance tasks due to their higher resistance to fatigue. Type II or fast-

twitch fibers assist more during fast, strength tasks due to their ability to achieve higher velocity contractions. Because untrained limb muscles have fiber type distributions similar to respiratory muscles, it is hypothesized that the expiratory muscles will adapt to strength training in the same way limb muscles do.

A recent endurance training study of respiratory muscles supports the above-mentioned hypothesis. Fourteen participants with chronic obstructive pulmonary disease completed a five-week endurance inspiratory muscle training program (Ramirez-Sarmiento et al., 2002). Biopsies of the external intercostal muscles at baseline and following training revealed a 38% increase in the number of type I fibers. This percent increase is similar to the changes found following endurance training of the limb muscles (Demirel et al., 2000) and provides evidence that respiratory muscles adapt to training much in the same manner as limb muscles.

The structural adaptations that occur in the respiratory muscles following training have also been studied by exposing rats to endurance type tasks. An increase in the number of mitochondria, capillary density, and type I muscle fibers have been found in rat diaphragmatic muscle tissue following inspiratory training (Keens et al., 1978; Powers et al., 1997). These adaptations in both human and animal respiratory muscles in response to endurance training are consistent with adaptations most frequently noted in limb muscles following endurance training programs (Holloszy & Coyle, 1984).

While the current research in both human and animal models has examined alterations in inspiratory muscles with endurance training, it is hypothesized that the expiratory muscles will respond to strength training in the same way. And while several studies have examined expiratory muscle training in both healthy and clinical populations

(Table 4-1) in regards to training load, duration of training, and training technique, there is great variability among all these factors and likewise, great variability in strength increases. The potential significance of the variability in these factors is described below.

Duration of training is an important consideration in the development of treatment programs as several factors influence a change in muscle strength over time. Changes in muscle strength of previously untrained limb muscles follow a pattern of large increases during approximately the first four weeks of training with much slower increases thereafter due to the significant contribution of neural adaptations during the initial stages of training (Moritani & deVries, 1979). Other investigations of neural contributions to strength increases have noted similar timelines (McComas, 1995; Sale, 1988). These neural adaptations include increased motor unit recruitment and better synchronization of motor unit firing resulting in stronger, more efficient power output from the muscle. Increases in strength beyond this period are generally due to hypertrophy. Due to similarities in structural adaptations to training in respiratory and limb muscles, it is assumed that respiratory muscles would follow a similar pattern of neural adaptations followed by hypertrophy.

Unfortunately, the majority of EMST studies reported in Table 4-1 have been carried out for six weeks or less. Therefore, it is difficult to make conclusions regarding the contribution of neural adaptations and hypertrophy to strength increase over time. The few studies that examined EMST for durations up to 12 weeks did not measure muscle strength on a weekly basis in order to track the pattern of change (Gosselink et al., 2000; Smeltzer et al., 1996). Understanding the pattern of increase in expiratory muscle strength in response to training has important implications for enhancing the clinical

applications of these techniques. For example, expiratory muscles may begin to plateau in regards to large increases in strength much later than four weeks, indicating the need for longer training periods to achieve a maximal training effect.

Another factor that may influence the achievement of maximal training effects is the incorporation of known effective training principles utilized extensively in limb strength training. Two such principles of strength training are: A) training at near maximal loads (overload), B) progressive increases in training loads over time (DeLorme, 1948). The overload principle states that a muscle must be exposed to exercise beyond the level to which it is accustomed in order to increase strength. Table 4-1 demonstrates that only two of the current EMST studies used training loads that would be considered near maximal (75%) (Hoffman Ruddy et al., 2001; Sapienza et al., 2002). The use of training at near maximal loads in these studies yielded higher strength increases than the other studies reported. Additionally, the amount of load employed in the training must be progressively increased over time in order to accommodate for the increasing strength of the muscle. Table 4-1 demonstrates that only three studies increased the training load throughout the training program. The remaining studies set the training load at the beginning of the study and did not alter the load to accommodate the increasing strength of the expiratory muscles. Again, the protocols that did increase the training load yielded higher strength increases than those that did not (Hoffman Ruddy et al., 2001; Sapienza et al., 2002).

The training techniques utilized in the studies described in Table 4-1 include pressure threshold training and resistance training. Pressure threshold training is a technique in which an individual must generate a minimal amount of pressure when

breathing through a training device to open a one-way valve. During resistance training, an individual breathes through a cylinder with a restricted orifice which provides resistance to the breath. During resistance training, the expiratory muscles activate to overcome an external resistance to flow. However, the expiratory muscles compensate for the resistance by slowing breathing rates and lowering airflow rates (Powers et al., 1997). This altered breathing pattern is an excellent compensation for the increased resistance, but unfortunately spares the expiratory muscles from high workloads thus reducing the strength training effect. During pressure threshold training, the individual must generate a constant minimum amount of force throughout the breath in order to open a spring-loaded one-way valve located in the training device. This results in greater force stress on the expiratory muscles. Therefore, pressure threshold training allows for more specificity to the ventilation maneuvers typically performed by the expiratory muscles than resistance-based training (Kellerman et al., 2000). It is difficult to determine the difference between the two techniques in regards to potential strength increase due to the small number of resistance studies and vaguely reported results. However, it is hypothesized that greater training effects would occur with pressure threshold training due to the mechanics of the technique.

The purpose of the current study was to examine the response of expiratory muscles to a training program of sufficient length (eight weeks) allowing for the examination of strength pattern increases across time. A training program was designed, incorporating known effective training principles from the limb muscle literature including overloading the targeted muscles and progressively increasing the training load to accommodate increases in strength. The training program also utilized pressure

threshold training due to its hypothesized potential for providing a greater training effect than resistance training. It was further hypothesized that the expiratory muscles would follow a similar pattern in their response to strength training as limb muscles with a large increase in strength found during the first four weeks of training, and the beginning of a plateau in strength thereafter.

Methods

Participants

Sixteen healthy participants completed this study. Six participants were males. The age of the males ranged from 19 to 32 years with an average age of 25.6 years. Ten participants were females. The age of the females ranged from 18 to 48 with an average age of 28.4 years. Participants were recruited from the Gainesville, Florida, area.

All participants had maximum expiratory pressure (MEP) values within a normative range for age and sex (Black & Hyatt, 1966). Pulmonary function testing was performed as a screening measure. All participants' forced expiratory volumes in the first second (FEV1) and forced vital capacity (FVC) measures were within normal limits (American Thoracic Society, 1987). See Table 4-2. All participants had a negative history of chronic and acute cardiac disease, upper respiratory infection, pulmonary dysfunction/disease, neuromuscular and immune system disease, vocal disturbances (chronic or acute), obesity, and no history of smoking within last 5 years (tobacco or recreational drugs).

Only those participants who were able to maintain their current level of physical activity (including both aerobic exercise and weightlifting) throughout the entire training period were included. Participants were specifically asked to report any significant

changes in their level of activity throughout their participation in the study with regards to intensity and frequency of exercise. An example of a significant change would entail a sedentary person beginning to exercise two to four days per week. Participants were discontinued in the study if they made a significant change in activity level as described above. Extreme athletes such as marathon runners and competitive weightlifters were also excluded.

Expiratory Muscle Strength

Expiratory muscle strength was measured indirectly as the maximum expiratory pressure (MEP) at the mouth. The measurement apparatus consisted of a mouthpiece connected to a pressure manometer by 50 cm of 2 mm i.d. tubing with a 14-guage-needle air-leak. In order to measure MEP, the participants' nose was occluded with nose clips. After inhaling to total lung capacity, the participant placed his or her lips around the mouthpiece and blew out as forcefully as possible. Three repeated measures were taken with a one-minute rest between trials, until the measurements obtained were within 5% of each other. The average of these three values was used.

Training Protocol

Participants completed a pressure threshold expiratory training program. The participants performed expiratory breathing exercises with the pressure threshold trainer five days per week. The training session consisted of five sets of five breaths at a threshold set at 75% of the participants' MEP. The participant completed the training sessions throughout the week independently at home. The pressure threshold was increased each week of training to reflect 75% of their MEP. The participants trained for eight weeks. Attempts were made to have an equal number of male and female

participants as males typically demonstrate greater increases in skeletal muscle strength in response to strength training programs (Ivey et al., 2000; Lewis et al., 1986).

Compliance

Participant compliance was acknowledged through participant education on the use of the device, mid-week contact by the investigator, and completion of a training log. Participants were provided with written and verbal instructions for the use of the device.

Statistical Analyses

The primary statistical method that was used to examine treatment differences with respect to the change from baseline for MEP was a repeated measures analysis of variance (ANOVA). Planned simple contrasts at alpha'=0.05 were utilized to examine differences between MEP and the baseline measure across the weeks of training. Reverse Helmert contrasts were also utilized to examine significant differences between training weeks during the program. The interaction of sex with MEP was also analyzed.

Results

The means and standard deviations as well as percent above baseline for MEP by training week are in Table 4-3. Mean MEP for males and females is depicted in Figure 4-2. Mean MEP for all participants by week is depicted in Figure 4-3.

A significant main effect was found for the within-subject factor of week (baseline through Week 8) $F(4.48, 62.74) = 18.217, p < .05$ (degrees of freedom were adjusted using the Huynh-Feldt correction factor as the sphericity assumption was not met). Planned simple contrasts revealed all weeks were significantly different than the baseline. Reverse Helmert contrasts in which all weeks were compared to the previous

week revealed significant differences for all comparisons except between Week 3 and Week 2 (Table 4-4).

Trainer Limitations

Three of the male participants were able to generate a MEP above 200 cmH_20 before the completion of the training program. The trainer was set to reflect 75% of the participants' MEP obtained each week. The maximum pressure threshold setting on the trainer used for this study was 150 cmH_20. Those participants who achieved MEPs above 200 cmH_20 during the training program continued to train with the trainer set at 150 cmH_20, however, this was at a pressure threshold less than the required 75% of their MEP. So even though these participants had achieved a MEP greater than 200 cmH_20, the training thresholds were unable to be raised in order to protect the participant from any potential risks that could have occurred when generating expiratory pressures above 150 cmH_20 repeatedly. Table 4-5 presents data from only those male participants who were able to train at 75% of their MEP throughout the entire training program.

Compliance

Four out of the 16 enrolled participants exhibited some aspect of non-compliance with the training protocol. Three of these participants reported missing only one training day during the one of the training weeks. The other participants reported missing two training days during one of the training weeks.

Attrition

All of the 16 participants who completed the study and whose data is reported in this study were able to meet with the investigators each week of the training program. There were an additional three participants who were discontinued the study protocol and

their results are not reported in this article. Two of the participants were discontinued after the first two weeks of training as they were unable to attend weekly meetings with the investigators. The other participant suffered an upper respiratory infection during at the fifth week of training and was unable to meet with the investigators.

Discussion

The results of this study revealed a significant increase in expiratory muscle strength with pressure threshold training. Maximum expiratory pressure increased by an average of 50% percent. This increase is comparable to other EMST training studies conducted with pressure-threshold training set at near maximal loads. The current study further supports that EMST is effective in increasing expiratory muscle strength.

It was hypothesized that the expiratory muscles would follow a similar pattern to limb muscles in regards to the pattern of strength increases. This hypothesis was based on the structural and functional similarities between expiratory and limb muscles and previous inspiratory muscle endurance training studies that revealed adaptations similar to those noted in limb muscles. Much of the limb literature suggests that large increases in strength due to neural adaptations are noted in approximately the first four weeks of training. Increases continue beyond this point, however, the increase begins to slow down or plateau somewhat. In this study, expiratory muscle strength increased rapidly during the first six weeks of training, after which a slower period of increase or the beginning of a plateau in strength was noted. Conclusions from this data indicate that expiratory muscles may have a longer period of neural adaptations than limb, but are similar. As there were only two data points following the Week 6 data point it is difficult to predict the trend of the data beyond this point. The data indicate that a longer period

of training (beyond eight weeks) is necessary to track the time course of strength increases. One interesting finding was that MEP did not increase significantly from Week 2 to Week 3. There are no hypotheses surrounding this lack of change during a training period typically characterized by rapid increases in strength.

These findings have important implications for future EMST protocol development in that participants may enroll in training programs longer than four weeks to gain full training effects. It also indicates that strength increases at six weeks are still primarily due to neural adaptations. Therefore, while a rapid increase in strength occurs in the first six weeks, cellular changes and hypertrophy of the muscles fibers may be limited during this time. Longer training periods may be necessary for structural muscle adaptations. How long these training periods have to be for significant hypertrophy to occur is yet to be determined. Cellular changes as well as muscle fiber changes may prevent the loss of strength during a detraining period. Examining structural and cellular changes in limb muscles with training are commonly completed by examining muscle biopsies (i.e. from the leg extensor muscles). Muscle biopsy from respiratory muscles are much more limited due to their location and potential adverse side effects with biopsy (i.e. pneumothorax). The previously mentioned study by Ramirez-Sarmiento and colleagues (2002) is the only study known to the author that has examined structural changes with respiratory training via muscle biopsy. Therefore, while traditional mechanisms may not be utilized at this time to study adaptations to training in expiratory muscles, studying force output over time with training is important for patient education and motivation for clinical applications of this technique.

Unfortunately, the limitations of the trainer used in this study restricts the ability to study the time course of training in individuals who initiate the program with higher than normal expiratory muscle strength (primarily males). However, a similar pattern of strength increase was found when examining only those subjects who were able to train at 75% of their MEP for the entire training period.

One of the factors that needs to be considered from an experimental and treatment protocol development standpoint is compliance. It was mentioned previously that the participants were provided with written and verbal instructions, had a mid-week contact with one of the investigators in addition to their weekly meeting, and were asked to complete a training log. As reported, the participants in this study were highly compliant with only three subjects omitting a training day on one occasion each and one subject omitting a training day on two occasions. Compliance is certainly an issue that is difficult to control with any home-program therapy intervention. The investigators feel that many efforts were made in this study to control for compliance, however, greater control could be obtained with participants completing all training sessions in the presence of the investigator. This increased contact time, however, would place a larger time commitment and on these participants, which may result in increased attrition.

The benefits of the training program described in this study are that it produces relatively fast results. The training program is easy to learn and training sessions at home last less than 15 minutes. The fact that the training procedure is fairly simple is beneficial to individuals with cognitive impairments and requires little or no assistance from a caregiver. As most of the training is completed at home, travel time for office visits with a speech-language pathologist or other health professionals is reduced. A

commonly used high intensity program for speech rehabilitation for patients with Parkinson's disease is the Lee Silverman Voice Therapy program (Ramig et al., 2001). This program requires the patient to attend therapy for four days a week for four weeks. The time commitment for EMST compared to this program is considerably less. Due to less travel time and visits with a health care professional, EMST may prove to be a more cost-effective rehabilitation technique. These aspects of the program may lend to greater patient motivation and compliance compared to other therapy techniques. Comparison of these two techniques would be of interest in patients with Parkinson's disease.

Future study of this training program should include tracking of strength changes beyond eight weeks of training to better understand the time course of the contribution of neural adaptations as well as the time to achieve the maximal training effect. Investigations of the program should also focus on studying the relationship between training frequency and intensity loads as variations in these parameters may alter the training effect.

Tables

Table 4-1. Summary of expiratory muscle strength training (EMST) study outcomes.

Population	Technique	Weeks	Training load	Load increased	Increase in MEP
Healthy[a]	PT	4	30% of MEP	No	25%
Healthy[b]	PT	2	75% of MEP	Yes	47%
Healthy[c]	PT	4	75% of MEP	Yes	84%
Mutliple[d] Sclerosis	PT	12	Not reported	No	37%
Multiple[e] Sclerosis	PT	12	60% of MEP	No	35%
Healthy[f]	RT	4	30% of MEP	No	n/a
Hypertonic[g] children	RT	6	2.5 cmH_20 to 7.5 cmH_20	Yes	69%

Note. PT = Pressure-threshold training; RT = Resistance training; MEP = maximum expiratory muscle strength.
[a](Suzuki et al., 1995). [b](Sapienza et al., 2002). [c](Hoffman Ruddy, et al., 2001). [d](Smeltzer et al., 1996). [e](Gosselink et al., 2000). [f](O'Kroy & Coast, 1993). [g](Cerny et al., 1997).

Table 4-2. Spirometry values for all participants.

	Mean	SD
FEV_1 (L/s)		
Females	2.874	.297
Males	3.774	.560
Average	3.211	.600
FVC (L/s)		
Females	3.417	.459
Males	4.398	.633
Average	3.785	.708

Note. FEV_1 = forced expiratory volume in one second; FVC = forced vital capacity.

Table 4-3. Maximum expiratory pressure all participants.

	Mean (cmH_20)	SD	Mean % above baseline
Baseline MEP			
Females	78.794	19.794	---
Males	132.845	26.687	---
Average	99.063	34.682	---
Week 1			
Females	101.910	34.141	29.33
Males	139.563	29.778	5.06
Average	116.030	36.734	17.12
Week 2			
Females	105.373	35.532	33.77
Males	145.520	27.917	9.54
Average	120.428	37.686	21.57
Week 3			
Females	111.259	42.041	41.20
Males	144.818	30.444	9.01
Average	123.843	40.632	25.01
Week 4			
Females	114.148	38.937	44.86
Males	149.717	37.301	19.26
Average	127.486	41.101	28.69
Week 5			
Females	121.584	35.646	54.31
Males	158.438	40.467	26.08
Average	136.326	40.745	37.62
Week 6			
Females	126.781	34.902	60.90
Males	167.492	40.713	27.21
Average	142.048	41.204	43.39
Week 7			
Females	122.360	33.880	55.29
Males	166.840	45.441	25.59
Average	139.040	43.262	40.36
Week 8			
Females	132.174	39.021	67.74
Males	175.072	45.858	31.78
Average	148.260	45.548	49.66

Table 4-4. Reverse Helmert contrasts for maximum expiratory pressure by week.

Source	df	Mean Square	F	p
Week 1 vs. Baseline	1	3259.830	8.162	.013*
Week 2 vs. Week 1	1	1610.403	12.858	.003*
Week 3 vs. Week 2	1	1480.548	4.505	.054
Week 4 vs. Week 3	1	1964.903	10.258	.007*
Week 5 vs. Week 4	1	5311.949	34.482	.000*
Week 6 vs. Week 5	1	7744.192	24.979	.000*
Week 7 vs. Week 6	1	3592.758	15.782	.002*
Week 8 vs. Week 7	1	7933.752	20.982	.001*

Note. $*p<.05$.

Table 4-5. Results for male participants who were able to train at 75% of their maximum expiratory pressure (MEP).

	Mean (cmH_20)	SD	Mean % above baseline
Baseline MEP			
Females	78.794	19.794	---
Males	126.393	29.877	---
Average	92.391	31.231	---
MEP after 8 weeks of training			
Females	132.174	39.021	70.80
Males	154.925	42.966	25.25
Average	138.672	39.922	57.79

Figures

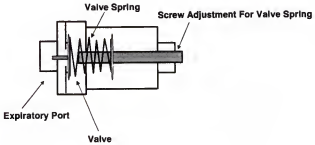

Figure 4-1. Schematic drawing of the expiratory pressure threshold trainer.

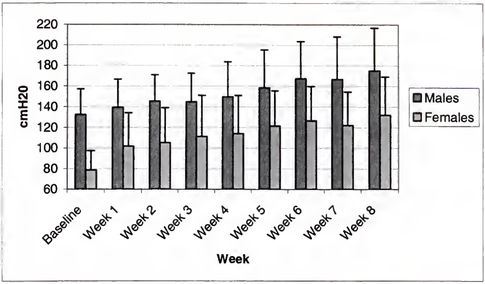

Figure 4-2. Mean maximum expiratory pressure by training week for males and females.

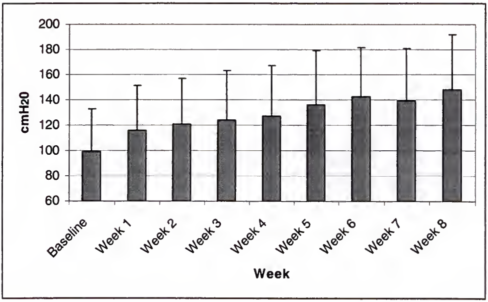

Figure 4-3. Mean maximum expiratory pressure by training week.

CHAPTER 5
DETRAINING OF EXPIRATORY MUSCLES

Introduction

Expiratory muscle strength training (EMST) programs demonstrate the ability to increase the strength of the expiratory muscles (Cerny et al., 1997; Gosselink et al., 2000; Hoffman Ruddy et al., 2001; O'Kroy & Coast, 1993; Sapienza et al., 2002; Smeltzer et al., 1996; Suzuki et al., 1995). Strength increases have ranged from 25 to 69% percent following four to eight weeks of training. Individuals whose expiratory muscle strength is compromised due to disease may benefit from improvements in expiratory muscle function. In fact, preliminary results of several studies utilizing EMST point to improvements in speech production (Cerny et al., 1997; Hoffman Ruddy et al., 2001) as well as cough production (Gosselink et al., 2000; Smeltzer et al., 1996).

Currently, there is little information about the stability of the strength increase following the removal of the training stimulus. Detraining in skeletal muscles is defined as a decrease in the adaptations that occur to a muscle as a result of training. The reduction of these adaptations results in eventual loss of maximum force output and therefore a loss in the functions that improve with the increased force output. Respiratory and limb muscles are structurally and functionally similar (Powers et al., 1997) and because of this, it might be assumed that respiratory muscles would follow a similar pattern of strength decline as limb muscles during a detraining period. The time course

of the reversibility of training effects gained from these programs is important for the development of effective training programs as well as patient education and motivation.

Human limb muscle research indicates that strengthened limb muscles (both leg and arm muscles) are able to maintain near maximal force generating ability for up to two to four weeks after the cessation of a training program (Coyle et al., 1984; Dudley et al., 1991; Hortobayi et al., 1993; Mujika & Padilla, 2001). Longer periods of detraining demonstrate more significant decreases in limb muscle strength, however, very minimal losses are generally seen even after two months of detraining. In a study of knee extensor strengthening in healthy males and females, Hakkinen and colleagues (1981; 1983) report only a 10 to 12% loss in strength following two months of detraining Diminished effects during more prolonged periods of detraining may include decreases in maximal force and reductions in muscle cross-sectional area related to a loss of contractile proteins (actin and myosinas well as neural adaptations (i.e., decrease in motor unit recruitment, decrease in synchronization of motor unit firing and coordination) related to inactivity (Hakkinen et al., 1981; 1983).

Only two respiratory muscle training studies address the concept of detraining following a strength training program. In a recent study, Romer and McConnell (2003) examined detraining following an inspiratory muscle pressure threshold training program in healthy participants. These participants completed a nine-week training program and were followed for 18 weeks after the completion of the training segment. The largest decrease in strength occurred during the first nine weeks of the detraining period which was only a 7% loss. Trends were not followed between the first and ninth week of the detraining period. No decreases in strength were noted between nine and 18 weeks of the

detraining period. As stated previously, the limb muscle literature suggests that training effects last up to four weeks. Additional data points during the first nine weeks of detraining period might have provided more specific information regarding the time course of strength loss.

Gosselink and colleagues (2000) examined detraining effects following an EMST program completed with nine individuals with multiple sclerosis. These participants completed a 12-week training program in which they increased their expiratory muscle strength by 35% from baseline. The participants were followed for six months following the program. The participants were able to maintain 30% of their strength above baseline at three-months following training. They maintained 9% of their strength above baseline six months following training. Information regarding the week-to-week time course of strength loss is unknown for these participants.

The effects of detraining are critically important to examine as the termination of a therapeutic program may result in a decline in performance and a possible return to baseline function. The effects of detraining, if they occur rapidly, can influence an individual's motivation to train and deter generalization of treatment to functional activities. Data about detraining trends following EMST programs can lend critical information for future development of treatment maintenance schedules.

The two studies described above suggest that a certain degree of detraining exists in respiratory muscles training, however, the purpose of this study was to further examine detraining effects following an EMST training program. It is hypothesized that expiratory muscles will follow a similar pattern as limb muscles during a detraining period in that the expiratory muscles will maintain the strength gained during the training

program for a period of approximately four weeks following training and will gradually decline over an additional four weeks of detraining. It is also hypothesized that similar detraining rates found in limb muscles (approximately 10-12% decrease) will be demonstrated in expiratory muscles. In an effort to specifically address the effect of detraining as a function of time, two groups of participants were enrolled in which one group trained for a longer period than the other. It was hypothesized that the group that trained for a longer period of time would have more adaptations to the expiratory muscles resulting in less loss of strength during the detraining period.

Methods

Participants

Thirty-two healthy participants completed this study. Twelve participants were males. The age of the males ranged from 18 to 32 years with an average age of 24.6 years. Twenty participants were females. The age of the females ranged from 19 to 48 years with an average age of 25.6 years Participants were recruited from the Gainesville, Florida, area.

All participants' MEP values were within a normative range for age and sex (Black & Hyatt, 1969). Pulmonary function testing was performed as a screening measure. All participants' forced expiratory volumes in the first second (FEV_1) and forced vital capacity (FVC) measures were within normal limits (American Thoracic Society, 1987). See Table 5-1. All participants had a negative history for chronic and acute cardiac disease including hypertension, upper respiratory infection, pulmonary dysfunction/disease, neuromuscular and immune system disease, vocal disturbances

(chronic or acute), obesity, and smoking within last five years (tobacco or recreational drugs).

Only those participants who were able to maintain their current level of physical activity (including both aerobic exercise and weightlifting) throughout the entire training period were recruited. In fact, participants were asked to report any significant changes in their level of activity throughout their participation in the study with regards to intensity and frequency of exercise (e.g. a sedentary person begins exercising two to four days per week). Participants were discontinued in the study if they made a significant change in activity level as described above. Extreme athletes such as marathon runners and competitive weightlifters were also excluded.

Measure of Expiratory Muscle Strength

Expiratory muscle strength was measured indirectly as the maximum expiratory pressure (MEP) at the mouth. The measurement apparatus consisted of a mouthpiece connected to a pressure manometer by 50 cm of 2 mm i.d. tubing with a 14-guage-needle air-leak. In order to measure MEP, the participants' occluded their nose with nose clips. After inhaling to total lung capacity, the participant placed his or her lips around the mouthpiece and blew out as forcefully as possible. Repeated measures were taken with a one-minute rest between trials, until three measurements were obtained within 5% of each other. The average of these three values was recorded.

Training Procedures

Expiratory pressure threshold trainer

An expiratory pressure threshold trainer was used to complete the expiratory muscle-training program. The trainer is a cylindrical device that consists of a mouthpiece

and a one-way spring-loaded valve (Figure 5-1). The valve blocks expiratory airflow until a sufficient threshold pressure is reached to overcome the spring force. To achieve this threshold pressure, the participant breathed out with an increased expiratory effort. As long as the threshold pressure was maintained, air flowed through the device. The device contains an adjustable spring, which allowed the required threshold pressure to be increased. The participants' MEP was measured at the initiation of the study and at the beginning of each subsequent training week. The threshold pressure was set at 75% of the participants' MEP at the time of measurement. Each training breath lasted two to three seconds.

Training protocol

Each participant was assigned to one of two training groups: a four-week training group or an eight-week training group. Each of the groups will be referred to as Group I or Group II, respectively. The participants in each of the two groups were matched for sex due to expected differences in male and female strength gains. Males typically demonstrate greater increases in skeletal muscle strength in response to strength training programs (Ivey et al., 2000; Lewis et al., 1986). Greater increases in MEP were found in males compared to females in a previously described study that utilized pressure-threshold training in healthy participants (Sapienza et al., 2002).

Group I. Participants completed a pressure threshold expiratory training program for four weeks. The threshold pressure was set at 75% of the participants' MEP at the time of measurement. The participants performed expiratory breathing exercises with the pressure threshold trainer five days per week. The training session consisted of five sets of five breaths at a threshold set at 75% of the participants' MEP. Each breath lasted two

to three seconds. The participant completed the training sessions throughout the week independently at home. The pressure threshold was increased each week of training to reflect 75% of their MEP.

Group II. Participants completed the same program described for Group I, however, they trained for a total of eight weeks.

Detraining. All participants were followed during an eight-week detraining period in which the expiratory muscle strength training program was discontinued. Participants were also asked to maintain their current activity level during this eight-week detraining period. Maximum expiratory pressure was measured at the end of the fourth week of detraining and at the eighth (final) week of the detraining period. Maximum expiratory pressure was not obtained each week as it represents a maximal force output maneuver, which might assist in maintaining strength if repeated frequently.

Compliance

Participant compliance was acknowledged through participant education on the use of the device, mid-week contact by the investigator, and completion of a training log. Participants were provided with written and verbal instructions for the use of the device.

Statistical Analyses

The primary statistical method that was used to examine treatment differences with respect to the change from baseline scores across the treatment groups for MEP was a repeated measures an analysis of variance (ANOVA). Week was the within-subject factor and sex was the between-subject factor. Significant differences at alpha'=0.05 were tested using planned simple contrasts. The interaction of sex with week was

analyzed as well. Paired t-tests were performed to compare Group I and II at the fourth and eighth week of detraining.

Results

Means and standard deviations as well as percent above baseline for MEP across the training and detraining periods are in Tables 5-2 and 5-3 and Figures 5-2 and 5-3. A significant main effect was found for the within-subject of week for both Group I and II (Table 5-4). Given the significant effect for both of these groups, analyzing simple contrasts further helped interpret the effect. Contrasts demonstrated that baseline MEP was significantly different at the final week of training for both Group I and II. As well, significant differences between baseline MEP and MEP at the end of the fourth week and eighth week of the detraining period were found for both groups. (Table 5-5). A main effect for the between-subject factor of sex was indicated for both Group I $F(1, 15) = 19.820$, $p < .05$, and Group II $F(1, 15) = 5.652$, $p < .05$.

Paired t-tests were used to compare the percent decrease from the final training week to both the four and eight week detraining points for Group I and II. Maximum expiratory pressure values were normalized by using the percent change from the final week of training. This normalization was performed to account for initial strength differences between the groups, which might contribute to an unbalance in MEP values at the final week of training. These analyses revealed that no significant difference existed between Group I and II at either the fourth week of detraining $t(16) = .391$, $p > .05$, or the eighth week $t(16) = .971$, $p > .05$

Six of the male participants (three in Group I; three in Group II) achieved a MEP above 200 cmH20 before the completion of the training program. The trainer was set to

reflect 75% of the participants' MEP obtained each week. The maximum setting on the trainer utilized for this study was 150 cmH_20. Those participants who achieved MEPs above 200 cmH_20 during the training program continued to train with the trainer set at 150 cmH_20, however, this was at a pressure threshold less than 75% of their MEP. So even though these participants had achieved a MEP greater then 200 cmH_20, the training threshold were unable to be raised in order to protect the participants from any potential risk that could have occurred when generating expiratory pressures above 150 cmH_20 repeatedly. Tables 5-6 and 5-7 present data from only those male participants who were able to train at 75% of their MEP throughout the entire training program.

Discussion

The results of this study revealed a significant increase in expiratory muscle strength for both Group I and II. Group I increased their MEP by 41% and Group II increased their MEP by 50%. Similar increases have been noted in other expiratory pressure threshold training studies. The current study further supports that EMST is effective in increasing expiratory muscle strength. It also suggests that EMST has the potential to impact speech and cough production parameters due to the increased pressure generating capability of the expiratory muscles.

Two important findings resulted from this study. The first finding was that the strength gained following the EMST program remained significantly above the baseline strength even after eight weeks of detraining in both Group I and II. At least for healthy individuals exposed to EMST, it appears that strength gains are maintained even when the training stimulus is removed. Slight decreases in strength that did occur during the detraining were gradual and consistent throughout the detraining period. It appears that

the expiratory muscles do follow a similar magnitude of decline in strength compared to limb muscles. The average decrease in strength during the detraining period in this study was 9%. As stated previously, Hakkinen and colleagues (1981; 1983) found a 10-12 % decrease in strength over an eight week detraining period in limb muscles. How detraining effects are affected by disease has yet to be determined. It is likely that detraining will occur sooner in populations where muscle structure or muscle physiology is compromised due to a decreased ability to adapt to exercise. It is apparent that a longer time course of study for the post-training period is necessary in order to determine when significant decreases in strength occur as well as to examine the interaction of disease state with detraining effect. Defining the time course of strength and the occurrence of detraining is important for future protocol development and for patient motivation and education

The second finding was that the two training groups did not differ significantly in the loss of strength. Regardless of whether training occurred for four or eight weeks, the degree of detraining was similar. It was hypothesized that Group II would demonstrate significantly less decline in strength during the detraining period. Longer training lengths allow for more structural adaptations to the muscles that might prevent detraining rates (i.e. hypertrophy, increase in contractile proteins). However, eight weeks of training may have been insufficient for noting structural adaptations that prevent detraining effects. In fact, Hakkinen and colleagues (1981) found only small amounts of hypertrophy in leg extensor muscles undergoing strength training at eight weeks compared to more significant hypertrophy at two months of detraining. Further work by Hakkinen and Komi (1983), which examined neuromuscular changes with detraining

suggests that the decrease in neural adaptations contributes most significantly to strength losses in the early stages of detraining (first eight weeks). Perhaps neural adaptations in Group I and II were fairly similar with the training program used in this study resulting in similar detraining rates.

The study of maintaining strength is certainly a logical next step for this line of research. In fact, Romer and Mcconnell (2003) addressed the issue of maintenance in the inspiratory muscle training study described previously. While one group of the participants completely ceased the training program, another group completed the program at two-thirds the training frequency compared to the training period. The participants in the maintenance group did not lose a significant amount of strength over the 18-week maintenance period from their final training week. Maintenance is an important concept for any strength training program and certainly warrants further study in expiratory muscle training.

The study of detraining in with other speech treatment techniques is fairly limited at this time. The majority of follow-up studies of maintenance or detraining have been conducted for behavioral programs for stuttering (Howie et al., 1981; Ingham et al., 2001; Ladoucer et al., 1982). A two-year follow-up study of speech therapy was conducted with patients who completed the Lee Silverman Voice Therapy (LSVT) program (Ramig et al., 2001). This therapy program is intended for patients with Parkinson's disease who benefit from increasing the amount of effort used for speech production. This program consists of encouraging patients to increase respiratory drive, laryngeal adduction, and articulatory motion during speech production. Improvements in speech production are thought to be due to neural reprogramming, increased coordination, and strengthening of

muscles groups used for speech production. The patients in the follow-up study only lost about 8% of their vocal intensity level (SPL) during reading and about 4% of their standard deviation of semitones (indicates range of vocal inflection) two years post treatment. Minimal losses such as those found in this study can be highly motivating to patients completing similar therapy programs. At this time, declines in training effects for EMST are only known out to two months for the protocol used in this study. The participants in both training groups only lost an average of 9% of their expiratory muscle strength following training. However, detraining in EMST is much different than in LSVT in that during the detraining period in the current study, the training stimulus was completely removed. For patients completing the LSVT program, it is expected that they will continue to use the "learned" higher, energy speaking pattern used in LSVT even after therapy is completed. The continuation of these speaking patterns, should maintain much of the gains achieved in therapy and it is suspected that LSVT detraining rates will be much lower than those noted in EMST over time.

One of the factors that needs to be considered from an experimental and treatment protocol development standpoint is participant compliance. It was mentioned previously that the participants were provided with written and verbal instructions, had a mid-week contact with one of the investigators in addition to their weekly meeting, and were asked to complete a training log. Compliance is certainly an issue that is difficult to control with any home-program therapy intervention, however, the investigators feel that many efforts were made in this study to control for compliance. It should be noted that the participants in this study were highly compliant with only three subjects omitting training days on one occasion and one participant omitting two training days during a training

week. The fact that the participants did not demonstrate a significant loss of strength during the detraining period provides further evidence that a stable training effect was achieved during the training. This stable training effect might not have been found without an acceptable level of compliance. Additionally, a similar training effect was found in healthy participants completing a four-week inspiratory pressure threshold training program (59% increase from baseline) in which all training was completed in the presence of one of the investigators.

A limitation of this training program is the pressure threshold limit on the trainer. Unfortunately, while this limit may reduce the ability to study individuals that initiate the program with high expiratory muscle strength, the pressure threshold cannot be raised due to safety concerns of generating pressures over 150 cmH_20 repeatedly.

The benefits of the training program described in this study are that it produces relatively fast results. The training program is easy to learn and training sessions at home only last less than 15 minutes. These aspects of the program may lend to greater patient motivation and compliance compared to other therapy techniques.

As EMST becomes implicated for use in clinical populations such as individuals with neuromuscular disease or spinal cord injury, the continued knowledge of detraining of these muscles will become necessary. Important aspects of muscle physiology in many neuromuscular disease processes are decreased muscle fiber size and poor innervation of the muscle. These characteristics may limit training effects with EMST and thus result in poor maintenance of effects once the training stimulus is removed. Detraining is generally a poorly studied concept in most therapy techniques. This lack of attention of detraining is unfortunate as maintaining training effects can be highly

motivating for patients and is frequently a query from patients regarding their course of treatment.

Tables

Table 5-1. Spirometry values for all participants.

	Mean	SD
FEV_1 (L/s)		
Females	3.050	.399
Males	4.095	1.055
Average	3.442	.870
FVC (L/s)		
Females	3.527	.473
Males	4.789	1.054
Average	4.000	.957

Note. FEV_1 = forced expiratory volume in one second; FVC = forced vital capacity.

Table 5-2. Maximum expiratory pressure (MEP) for all participants in Group I.

	Mean (cmH_20)	SD	Mean % above baseline
Baseline MEP			
Females	83.343	19.402	---
Males	140.600	29.174	---
Average	104.814	36.457	---
MEP after four weeks of training			
Females	123.353	24.527	48.00
Males	187.543	35.811	33.38
Average	147.424	42.644	40.65
Four weeks post-training			
Females	119.057	20.708	42.85
Males	177.150	46.881	25.99
Average	140.842	42.821	34.37
Eight weeks post-training			
Females	114.118	20.594	36.93
Males	173.810	37.580	23.62
Average	136.503	40.200	30.23

Table 5-3. Maximum expiratory pressure (MEP) for all participants in Group II.

	Mean (cmH_2O)	SD	Mean % above baseline
Baseline MEP			
Females	78.794	19.794	---
Males	132.845	26.687	---
Average	99.063	34.682	---
MEP after eight weeks of training			
Females	132.174	39.021	67.74
Males	175.072	45.858	31.79
Average	148.261	45.547	49.66
Four weeks post-training			
Females	125.023	34.982	58.67
Males	164.256	50.031	26.38
Average	139.735	44.197	41.05
Eight weeks post-training			
Female	118.908	40.889	50.91
Male	167.898	48.394	26.39
Average	135.238	48.118	36.52

Table 5-4. Results of the repeated measures ANOVA examining the effect of weeks of training on all dependent variables

Training Group	Source	df	F	*p*
Group I	Week	4.059	17.058	.000*
Group II	Week	4.224	13.789	.000*

Note. $*p<.05$.

Table 5-5. Planned simple contrasts for maximum expiratory pressure by week.

Group	Source	df	Mean Square	F	*p*
I					
	Baseline vs. wk4	1	28353.308	43.653	.000*
	Baseline vs. D1	1	19582.821	22.761	.000*
	Baseline vs. D2	1	15352.801	22.783	.000*
II					
	Baseline vs. wk8	1	34778.641	31.769	.000*
	Baseline vs. D1	1	23655.306	18.561	.001*
	Baseline vs. D2	1	17263.512	11.262	.006*

Note. wk4 = fourth and final week of training; wk8 = eighth and final week of training; D1 = fourth week of detraining period; D2 = eighth and final week of detraining period. $*p<.05$.

Table 5-6. Results for male participants in Group I who were able to train at 75% of their maximum expiratory pressure.

	Mean (cmH_20)	SD	Mean % above baseline
Baseline MEP	133.500	34.567	---
Week 8	159.510	25.952	19.17
D1	136.700	23.995	2.39
D2	141.467	19.807	5.97

Note. D1 = fourth week of detraining period; D2 = eighth and final week of detraining period.

Table 5-7. Results for male participants in Group II who were able to train at 75% of their maximum expiratory pressure.

	Mean (cmH_20)	SD	Mean % above baseline
Baseline MEP	126.393	29.877	---
Week 8	154.915	42.966	25.25
D1	141.858	46.167	15.75
D2	142.077	44.489	17.33

Note. D1 = fourth week of detraining period; D2 = eighth and final week of detraining period.

Figures

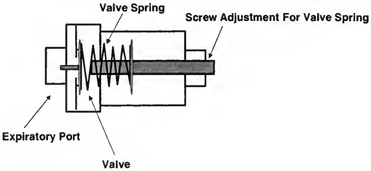

Figure 5-1. Schematic drawing of the expiratory pressure threshold trainer.

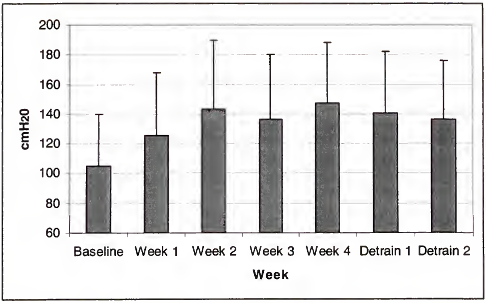

Figure 5-2. Mean maximum expiratory pressure across all training and detraining weeks for Group I.

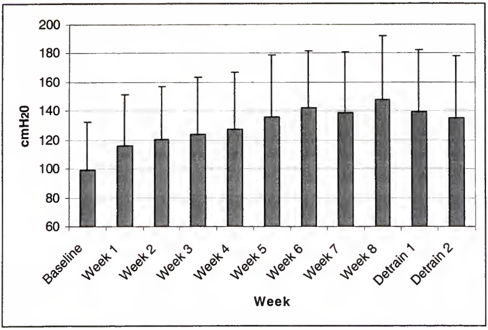

Figure 5-3. Mean maximum expiratory pressure across all training and detraining weeks for Group II.

CHAPTER 6
GENERAL DISCUSSION

It has been established in the current literature that EMST is effective in increasing expiratory muscle strength. However, many issues pertaining to the use of this technique as a treatment modality have not been addressed previously. These issues include effective training lengths, detraining of the expiratory muscles, and the translation of increase in expiratory muscle strength to physiologic alterations in speech and cough. Therefore, the purpose of the presented studies was to begin a methodological examination of this training technique with particular attention to the time course of training lengths and detraining. The focus of these studies was also to build on subjective reports of improvements in speech and cough and to examine the physiologic changes with expiratory muscle training.

The results of the examination of speech production with increased expiratory muscle strength revealed significant changes in the physiologic work used for the generation of subglottal pressure (P_s) and intraoral pressure (P_0). While certainly the physiologic work in the form of expiratory muscle activation for speech production in healthy individuals is fairly low, this is not likely the case for individuals with disease processes resulting in expiratory muscle weakness. Future investigations of perceived effort during speech tasks with increased expiratory muscle strength are of particular interest in individuals with expiratory muscle weakness and those who speak in high effort situations (speaking/singing while physically active). Other speech measures such

as maximum P_s and peak RMS did not reveal significant changes with EMST. While these parameters did not change in healthy individuals it should not be discounted that they may improve in individuals who have difficulty achieving louder speech due to expiratory muscle weakness.

The results of the examination of cough production revealed no significant change in maximum flow rate and rise time. There was, however, a trend noted in the compression time and in fact a significant effect was found for Group II. The time the glottis was closed prior to the explosive phase of the cough decreased by 22% with training in Group II. This change warrants further investigation and might be more pronounced in individuals with expiratory muscle weakness. This physiologic alteration has important implications for individuals with poor adduction of the vocal folds due to injury or age. Future investigations of this technique are recommended for individuals with laryngeal dysfunction to examine alterations in peak flows and compression time.

The results of the examination of training length revealed important information regarding when strength increases may begin to plateau. It appears that this may begin after six weeks of training, however, a longer training time would be necessary to determine the trend. Observing training week-by-week such as performed in this study, provides insight into the time-course of neural adaptations and cellular/structural adaptation to the expiratory muscles. Understanding this time course will allow investigators and clinicians to know the necessary training length to achieve maximal effects in strength and potentially in maximal effects in speech and cough improvement.

The result of the examination of detraining of the expiratory muscles revealed that the participants only lost an average of 9% of strength from the final measure at eight

weeks post-training. These findings suggest that a longer detraining period must be studied in order to determine when significant decreases begin. This information will facilitate patient counseling regarding therapy maintenance protocols. No significant difference in detraining magnitudes were noted between Group I and II which indicated the longer training times do not necessarily prevent detraining. The study of detraining in this methodological examination is somewhat unique in that detraining is a poorly studied concept in many therapeutic interventions. However, patients frequently express concerns over the results of discontinuing a training program. Therefore, an understanding of detraining is highly relevant to patient education and motivation.

Certainly as discussed in previous chapters, this training technique is not without limitations. The current model for training consists of independent training sessions that occur in the home. Efforts are made for participant compliance, however, at the current time, there is no assurance that the participant is being fully compliant. Additionally, participant attrition is a concern when studying humans over longer periods of time. Attrition in this study was fairly low, however, it has been suggested from these findings that longer time courses of training and detraining be investigated. More difficulty with attrition and non-compliance may be encountered in these studies.

The ease of using the device and relatively short training periods are among the benefits of this training program. The participants in this study were able to complete the training program in their home generally within 10 to 15 minutes for each training day. The low time investment may be beneficial in patient compliance for clinical implementation of this program.

While this study was completed with healthy subjects it provides a comparison point and a potential guideline for protocol development. The study of strengthening respiratory muscles is much more difficult than limb muscles, and therefore, the literature is not nearly as complete. Previously, training studies for respiratory muscles have somewhat followed models presented in limb muscles strengthening research.

Currently, EMST is being applied to clinical populations including individuals with Parkinson's disease, multiple sclerosis, and spinal cord injury. The investigation of the parameters evaluated in this study in individuals with expiratory muscle weakness may provide additional information regarding the effectiveness of this treatment technique.

APPENDIX

PAPA PASSAGE

Papa was a great man. Working all of his life as a carpenter, he built homes for other people. Papa was an excellent craftsman. Anyone who worked with Papa knew that he was an honest man. Papa gave himself to his work, toiling daily for small amounts of money. No one disliked Papa. In fact, neighbors used to bring Papa apples, pears, and other fruits, especially around the holidays. I remember Papa for his kind ways. What I remember was the manner in which Papa dressed, the way he carried himself. Papa was such a strong man. Devoted to his family, especially his children. Papa worked night and day to provide for us. Although we never showed Papa our appreciation on a daily basis, I know that he felt our love, or so I hope.

REFERENCE LIST

Addington, W.R., Stephens, R.E., & Gilliland, K.A. (1999). Assessing the laryngeal cough reflex and the risk of developing pneumonia after stroke. *Stroke, 30,* 1203-1207.

American Thoracic Society. (1987). Standardization of spirometry: 1987 update. *American Review of Respiratory Disorders, 136,* 1285-1298.

Arora, N.S., & Gal, T.J. (1981). Respiratory mechanics in supine subjects during progressive partial curarization. *Journal of Applied Physiology, 52,* 57-63.

Asher, M.I., Pardy, R.L., Coates, A.L., Thomas, E., & Macklem, P.T. (1982). The effects of inspiratory muscle training in patients with cystic fibrosis. *The American Review of Respiratory Disease, 126,* 855-859.

Bach, J.R., Smith, W.H., Saporito, L., Alba, A.S., Dayal, R., & Pan, J. (1993). Airway secretion clearance by mechanical exsufflation for post-poliomyelitis ventilator-assisted individuals. *Archives of Physical Medicine and Rehabilitation, 74,* 170-177.

Bailey, F., & Hoit, J. (2002). Speaking and breathing in high respiratory drive. *Journal of Speech, Language, and Hearing Research, 45,* 89-99.

Baker, S., Sapienza, C., & Collins, S. (2003). Inspiratory muscle training in pediatric patients with vocal fold paralysis. *International Journal of Pediatric Otorhinolaryngology, 67,* 413-416.

Baker, S., Sapienza, C., Davenport, P., Martin, D., Hoffman, B., Woodson, G. (in press). Inspiratory pressure threshold training for upper airway limitation: A case of bilateral abductor vocal fold paralysis. *Journal of Voice.*

Bartlett, D., Remmers, J., & Gautier, H. (1973). Laryngeal regulation of respiratory airflow. *Respiratory Physiology, 18,* 194-204.

Black, L.F., & Hyatt, R.E. (1969). Maximal respiratory pressures: Normal values and relationship to age and sex. *American Review of Respiratory Disease, 99,* 696-702.

Bouros, D., Siafakas, N., & Green, M. (1995). Cough: Physiological and pathophysiological considerations. *In* C. Roussos, editor. *The Thorax*, Vol. B. Marcel Dekker Inc., New York. 1335-1354.

Cerny, F., Panzarella, K., & Stathopoulus, E. (1997). Expiratory muscles conditioning in hyptonic children with low vocal intensity levels. *Journal of Medical Speech-Language Pathology, 5,* 141-152.

Chou, D., & Wang, S. (1975). Studies on the localization of central cough mechanism: site of action of antitussive drugs. *Journal of Pharmacology Experimental Therapy, 194,* 499-505.

Coyle, E., Martin, W., Sinacore, D., Joyner, M., Hagberg, J., & Holloszy, J. (1984). Time course of loss of adaptations after stopping prolonged intense endurance training. *Journal of Applied Physiology, 57,* 1857-1864.

Dawid-Milner, M., Lara, J., Milan, A., & Gonzalez-Baron, S. (1993). Activity of inspiratory neurons of the ambiguous complex during cough in the spontaneously breathing decerebrate cat. *Experimental Physiology, 78,* 835-838.

deJong, W., vanAalderen, W.M., Kraan, J., Koeter, G.H., & vanderSchans, C.P. (2001). Inspiratory muscle training in patients with cystic fibrosis. *Respiratory Medicine, 95,* 31-36.

DeLorme, T.L. (1948). Techniques of progressive resistance exercise. *Archives of Physical Medicine Rehabilitation, 29,* 263-273.

Demirel, H., Powers, S., Naito, H., Hughes, M. & Coombes, J. (2000). Exercise-induced alterations in skeletal muscle myosin heavy chain phenotype: Dose-response relationship. *Journal of Applied Physiology, 86,* 1002-1008.

Derrickson, J., Ciesla, N., Simpson, N., & Imle, P.C. (1992). A comparison of two breathing exercise programs for patients with quadriplegia. *Physical Therapy, 72,* 763-769.

DiMarco, A.F., Romaniuk, J.R., & Supinski, G.S. (1995). Electrical activation of the expiratory muscles to restore cough. *American Journal of Respiratory and Critical Care Medicine, 151,* 1466-1471.

Dudley, G.A., Tesch, P.A., Miller, B.J., & Buchanan, P. (1991). Importance of eccentric actions in performance adaptions to resistance training. *Aviation Space and Environmental Medicine, 62,* 543-550.

Gosselink, R., & Decramer, M. (1994). Inspiratory muscle training: Where are we? *European Respiratory Journal, 7,* 2103-2105.

Gosselink, R., Kovacs, L., Ketelar, P., Carton, H., & Decramer, M. (2000). Respiratory muscle weakness and respiratory muscle training in severely disabled multiple sclerosis patients. *Archives of Physical Medicine Rehabilitation, 81,* 747-751.

Grassino, A. (1989). Inspiratory muscle training in COPD patients. *European Respiratory Journal. Supplement, 7,* 581s-586s.

Hakkinen, K., & Komi, P. (1983). Electromyographic changes during strength training and detraining. *Medicine and Science in Sports and Exercise, 15,* 455-460.

Hakkinen, K., Komi, P., & Tesch, P. (1981). Effects of combined concentric and eccentric strength training and detraining on force-time, muscle fiber and metabolic characteristics of leg extensor muscles. *Scandanavian Journal of Sports Science, 3,* 50-58.

Hixon, T.J. (1973). Kinematics of the chest wall during speech production: Volume displacements of the rib cage, abdomen, and lung. *Journal of Speech and Hearing Research, 16,* 78-115.

Hixon, T.J. (1987). *Respiratory function in speech and song.* San Diego, CA: Singular Publishing Group, Inc.

Hixon, T.J., Mead, J., & Goldman, M.D. (1976). Dynamics of the chest wall during speech production: Function of the thorax, rib cage, diaphragm, and abdomen. *Journal of Speech and Hearing Research, 19,* 297-356.

Hixon, T.J., & Weismer, G. (1995). Perspectives on the Edinburgh study of speech breathing. *Journal of Speech and Hearing Research, 38,* 42-60.

Hoffman Ruddy, B., Sapienza, C., Davenport, P., Martin, D., & Lehman, J. (2001). *Expiratory pressure threshold training in "high risk" performers.* Poster Session. American Speech-Language-Hearing Association Annual Convention. New Orleans, Louisiana.

Holas, M.A., DePippo, K.L., & Reding, M.J. (1994). Aspiration and relative risk of medical complications following stroke. *Archives of Neurology, 51,* 1051-1053.

Holloszy, J.O., & Coyle, E.F. (1984). Adaptations of skeletal muscle to endurance exercise and their metabolic consequences. *Journal of Applied Physiology, 56,* 831-838.

Horner, J., Massey, E.W., Riski, J.E., Lathrop, D.L., & Chase, K.N. (1988). Aspiration following stroke: Clinical correlates and outcome. *Neurology, 38,* 1359-1362.

Hortobagyi, T., Houmard, J., Stevenson, J., Fraser, D., Johns, R., & Israel, R. (1993). The effects of detraining on power athletes. *Medicine and Science in Sports and Exercise,* 929-935.

Howie, P., Tanner, S., & Andrews, G. (1981). Short-and long-term outcome in an intensive treatment program for adult stutterers. *Journal of Speech and Hearing Disorders, 46,* 104-109.

Ingham,R., Kilgo, M., Ingham, J., Moglia, R., Belknap, H., & Sanchez, T. (2001). Evaluation of a stuttering treatment based on reduction of short phonation intervals. *Journal of Speech, Language, Hearing, and Research, 44,* 1229-1244.

Inkley, S.R., Oldenburg, F.C., & Vignos P.J. (1974). Pulmonary function in Duchenne muscular dystrophy related to stage of disease. *American Journal of Medicine, 56,* 297-306.

Isshiki, N. (1964). Regulatory mechanism of voice intensity variation. *Journal of Speech and Hearing Research, 7,* 17-29.

Ivey, F.M., Tracy, B.L., Lemmer, J.T., NessAiver, M., Metter, E.J., Fozard, J.L., & Hurley, B.F. (2000). Effects of strength training and detraining on muscle quality: Age and gender comparisons. *Journal of Gerontology, 55*, B152-B157.

Jacobson, B., Johnson, A., Grywalski, C., Silbergleit, A., Jacobson, G., & Benninger, M. (1997). The Voice Handicap Index (VHI): Development and Validation. *American Journal of Speech-Language Pathology, 6*, 66-70.

Jaeger, R.J., Turba, R.M., Yarkony, G.M., & Roth, E.J. (1993). Cough in spinal cord injured patients: comparison of three methods to produce cough. *Archives of Physical Medicine and Rehabilitation, 74,* 1358-1361.

Keens, T.G., Chen, V., Patel, P., O'Brien, P., Levison, H., & Ianuzzo, C.D. (1978). Cellular adaptations of the ventilatory muscles to a chronic increased respiratory load. *Journal of Applied Physiology, 44*, 905-908.

Kellerman, B., Davenport, P., & Martin, D. (2000). Inspiratory strengthening effect on resistive load detection and magnitude estimation. *Medicine and Science in Sports and Exercise, 32,* 1859-1867.

Ladouceur, R., Cote, C., Leblond, G., & Brouchard, L. (1982). Evaluation of regulated-breathing method and awareness training in treatment of stuttering. *Journal of Speech and Hearing Disorders, 47,* 422-426.

Langlands, J. (1967). The dynamics of cough in health and in chronic bronchitis. *Thorax, 22,* 88-96.

Lansing, R., Im, B., Thwing, J., Legedza, A., & Banzett, R. (2000). The perception of respiratory work and effort can be independent of the perception of air hunger. *American Journal of Respiratory and Critical Care Disease, 147,* 1199-1206.

Larson, J.L., Kim, M.J., Sharp, J.T., & Larson, D.A. (1988). Inspiratory muscle training with a pressure threshold breathing device in patients with chronic obstructive pulmonary disease. *American Review of Respiratory Disease, 138,* 689-696.

Leiner, G.C., Abramowitz, S., Small, M.J., & Stenby, V.B. (1966). Cough peak flow rate. *American Journal of the Medical Sciences, 251,* 211-214.

Leith, D.E., & Bradley, M.E. (1976). Ventilatory muscle strength and endurance training. *Journal of Applied Physiology, 41,* 508-516.

Lewis, D.A., Kamon, E., & J.L. Hodgson (1986). Physiological differences between genders: Implications for sports conditiong. *Sports Medicine, 3,* 357-369.

Lofqvist, A., Carlborg, B, & Kitzing, P. (1982). Initial validation of an indirect measure of subglottal pressuring during vowels. *Journal of the Acoustical Society of America, 72,* 633-635.

McComas, A. (1995). Human neuromuscular adaptations that accompany changes in activity. *Medicine and Science in Sports and Exercise, 26,* 1498-1509.

McCool, F.D., & Leith, D.E. (1987). Pathophysiology of cough. *Clinical Chest Medicine, 8,* 189-195.

Mitchell, H., Hoit, J., & Watson, P. (1996). Cognitive-linguistic demands and speech breathing. *Journal of Speech and Hearing Research, 39,* 93-104.

Mizuno, M. (1991). Human respiratory muscles: Fibre morphology and capillary supply. *European Respiratory Journal, 4,* 587-601.

Mujika, I., & Padilla, S. (2001). Muscular characteristics of detraining in humans. *Medicine and Science in Sports and Exercise, 33,* 1297-1303.

Moritani, T., & deVries, H. (1979). Neural factors versus hypertrophy in the time course of muscle strength gain. *American Journal of Physical Medicine, 58,* 115-130.

Netsell, R. (1969). Evaluation of velopharyngeal function in dysarthria. *Journal of Speech and Hearing Disorders, 34,* 113-122.

Netsell, R., & Hixon, T.J. (1978). A noninvasive method for clinically estimating subglottal air pressure. *Journal of Speech and Hearing Disorders, 43,* 326-330.

Nield, M.A. (1999). Inspiratory muscle training protocol using a pressure threshold device: Effect on dyspnea in chronic obstructive pulmonary disease. *Archives of Physical Medicine and Rehabilitation, 80,* 100-102.

O'Kroy, J.A., & Coast, J.R. (1993). Effects of flow and resistive training on respiratory muscle endurance and strength. *Respiration, 60,* 279-283.

Polkey, M.I., Lyall, R.A., Green, M., Nigel-Leigh, P., & Moxham, J. (1998). Expiratory muscle function in amyotrophic lateral sclerosis. *American Journal of Respiratory Critical Care Medicine, 158,* 734-741.

Powers, S., Coombes, J., & Demirel, H. (1997). Exercise training-induced changes in respiratory muscles. *Sports Medicine, 24,* 120-131.

Powers, S., & Howley, E. (2001). *Exercise physiology: Theory and application to fitness and performance* (4^{th} ed.). New York, NY: McGraw-Hill.

Ramig, L., Sapir, S., Countryman, S., Pawlas, A., O'Brien, C., Hoehn, M., & Thompson, L. (2001). Intensive voice treatment (LSVT) for patients with Parkinson's disease: A 2 year follow up. *Journal of Neurology, Neurosurgery, and Psychiatry, 71,* 493-498.

Ramig, L., Sapir, S., Fox, C., & Countryman, S. (2001). Changes in vocal loudness following intensive voice treatment (LSVT) in individuals with Parkinson's disease: A comparison with untreated patients and normal age-matched controls. *Movement Disorders, 16,* 79-83.

Ramirez-Sarmiento, A., Orozco-Levi, R., Barreiro, E., Hernandez, N., Mota, S., Sangenis, M., Broquetas, J., Casan, P., & Gea, J. (2002). Inspiratory muscle training in patients with chronic obstructive pulmonary disease: Structural adaptations and physiologic outcomes. *American Journal of Respiratory Critical Care Medicine, 166,* 1491-1497.

Romer, L., & McConnell, A. (2003). Specificity and reversibility of inspiratory muscle training. *Medicine and Science in Sports and Exercise, 35,* 237-244.

Roy, N., Weinrich, B., Gray, S., Tanner, K., Stemple, J., & Sapienza, C. (2003). *Journal of Speech, Language, and Hearing Research, 46,* 670-688.

Sale, D.G. (1988). Neural adaptation to resistance exercise. *Medicine and Science in Sports and Exercise, 20,* S135-S145.

Sant'Ambrogio, G., Mathew, O., Fisher, J., & Sant'Ambrogio, F. (1983). Laryngeal receptors responding to transmural pressure, airflow, and local muscle activity. *Respiratory Physiology, 54,* 317-330.

Sapienza, C., Brown, J., Martin, D., & Davenport, P. (1999). Inspiratory pressure threshold training for glottal airway limitation in laryngeal papilloma. *Journal of Voice, 13*, 382-388.

Sapienza, C., Davenport, P, & Martin, D. (2001). Pressure threshold training with a spinal cord injured patient. International Association for Logapedics and Phoniatrics. Montreal, Quebec.

Sapienza, C., Davenport, P, & Martin, D. (2002). Expiratory muscle training increases pressure support in high school band students. *Journal of Voice, 1,* 495-501.

Sapienza, C.M., & Stathopoulos, E.T. (1995). Speech task effects on acoustic and aerodynamic measures of women with vocal nodules. *Journal of Voice, 9*, 423-428.

Scherer, R. (1991). Physiology of phonation: a review of basic mechanics. In C.N. Ford and D.M. Bless (Eds.) *Phonosurgery: Assessment and Surgical Management of Voice Disorders*. New York, NY: Raven Press.

Schneider, P., & Baken, R. (1984). Influence of lung volume on the airflow-intensity relationship. *Journal of Speech and Hearing Research, 27*, 430-435.

Shaffer, T., Wolfson, M., & Bhutani, V. (1981). Respiratory muscle function, assessment, and training. *Physical Therapy, 61,* 1711-1723.

Shah, M.D., & Shah, S.M. (2001). The applied physiology of cough. *Indian Journal of Pediatrics, 68 Supplement 2,* S3-S10.

Simpson, L.S. (1983). Effect of increased abdominal muscle strength on forced vital capacity and forced expiratory volume. *Physical Therapy, 63*, 334-337.

Sivasothy, P., Brown, L., Smith, I.E., & Shneerson, J.M. (2001). Effects of manually assisted cough and mechanical insufflation on cough flow of normal subjects, patients with chronic obstructive pulmonary disease (COPD), and patients with respiratory muscle weakness. *Thorax, 56,* 438-444.

Smeltzer, S.C., Lavietes, M.H., & Cook, S.D. (1996). Expiratory training in multiple sclerosis. *Archives of physical medicine and rehabilitation, 77,* 909-912.

Smith-Hammond, C., Goldstein, L., Zajac, D., Gray, L., Davenport, P., & Bolser, D. (2001). Assessment of aspiration risk in stroke patients with quantification of voluntary cough. *Neurology, 56*, 502-506.

Smitheran, J. & Hixon, T. (1981). Method for estimating laryngeal resistance during vowel production. *Journal of Speech and Hearing Disorders, 46,* 138-146.

Solomon, N., & Hixon, T. (1993). Speech breathing in Parkinson's disease. *Journal of Speech and Hearing Research, 34,* 761-767.

Sonne, L.J, & Davis, J.A. (1982). Increased exercise performance in patients with severe COPD following inspiratory resistive training. *Chest, 81,* 436-439.

Suarez, A.A., Pessolano, F.A., Monteiro, S.G., Ferreyra, G., Capria, M.E., Mesa, L., Dubrovsky, A., & DeVito, E.L. (2002). Peak flow and peak cough flow in the evaluation of expiratory muscle weakness and bulbar impairment in patients with neuromuscular disease. *American Journal of Physical Medicine Rehabilitation, 81,* 506-511.

Suzuki, S., Sato, M., & Okubo, T. (1995). Expiratory muscle training and sensation of respiratory effort during exercise in normal subjects. *Thorax, 50,* 366-370.

Szeinberg, A., Tabachnik, E., Rashed, N., McLaughlin, F.J., England, S, Bryan, C.A., & Levison, H. (1988). Cough capacity in patients with muscular dystrophy. *Chest, 94,* 1232-1235.

Taylor, P., Tromans, A., Harris, K., & Swain, I. (2002). Electrical stimulation of abdominal muscles for control of blood pressure and augmentation of cough in a C3/4 level tetraplegic. *Spinal Cord, 40,* 34-36.

Titze, I.R. (1994). *Vocal Fold Physiology.* San Diego, California: Singular Publishing Group, Inc.

Vegeret, J., Kays, C., Choukroun, M.L., Douvier, J.J., Taytard, A., & Guenard, H. (1987). Expiratory muscles and exercise limitations in patients with chronic obstructive pulmonary disease. *Respiration, 52,* 181-188.

Weiner, P., Azgad, Y., Ganam, R., & Weiner, M. (1992). Inspiratory muscle training in patients with bronchial asthma. *Chest, 102,* 1357-1361.

Weiner, P., Berar-Yanay, N., Davidovich, A., Magadle, R., & Weiner, M. (2000). Specific inspiratory muscle training in patients with mild asthma with high consumption of inhaled beta(2)-agonists. *Chest, 117,* 722-727.

Weins, M.E., Reimer, M.A., & Guyn, H.L. (1999). Music therapy as a treatment method for improving respiratory strength in patients with advanced multiple sclerosis: A pilot study. *Rehabilitation Nursing, 24,* 74-80.

West, J. (2000). Respiratory physiology: *The essentials.* (5th ed.). Baltimore, MD: Williams & Wilkins.

Widdicombe, J. (1982). Pulmonary and respiratory tract receptors. *Journal of Experimental Biology, 100,* 41-57.

Widdicombe, J. (1995). Neurophysiology of the cough reflex. *European Journal of Respiratory Physiology, 8,* 1193-1202.

Widdicombe, J. (2001). Airway receptors. *Respiration Physiology, 125,* 3-15.

BIOGRAPHICAL SKETCH

Susan E. Baker received a Bachelor of Science in 1997 and a Master of Arts in 1999 from Miami University. In August 2000 she enrolled in the doctoral program in the Department of Communication Sciences and Disorders at the University of Florida. Her research during her doctoral program has focused on investigating the role of expiratory muscle strength training for the improvement of voice and cough production. She will graduate from the University of Florida with a Doctorate of Philosophy in August of 2003 after which she will begin a position as an assistant professor at Miami University.

I certify that I have read this study and that in my opinion it conforms to acceptable standards of scholarly presentation and is fully adequate, in scope and quality, as a dissertation for the degree of Doctor of Philosophy.

Christine M. Sapienza, Chair
Associate Professor of Communication Sciences and Disorders

I certify that I have read this study and that in my opinion it conforms to acceptable standards of scholarly presentation and is fully adequate, in scope and quality, as a dissertation for the degree of Doctor of Philosophy.

William S. Brown, Jr.
Professor of Communication Sciences and Disorders

I certify that I have read this study and that in my opinion it conforms to acceptable standards of scholarly presentation and is fully adequate, in scope and quality, as a dissertation for the degree of Doctor of Philosophy.

Howard B. Rothman
Professor of Communication Sciences and Disorders

I certify that I have read this study and that in my opinion it conforms to acceptable standards of scholarly presentation and is fully adequate, in scope and quality, as a dissertation for the degree of Doctor of Philosophy.

Paul Davenport
Professor of Veterinary Medicine

This dissertation was submitted to the Graduate Faculty of the Department of Communication Sciences and Disorders in the College of Liberal Arts and Sciences and the Graduate School and was accepted as partial fulfillment of the requirements for the degree of Doctor of Philosophy.

August 2003

Dean, Graduate School